

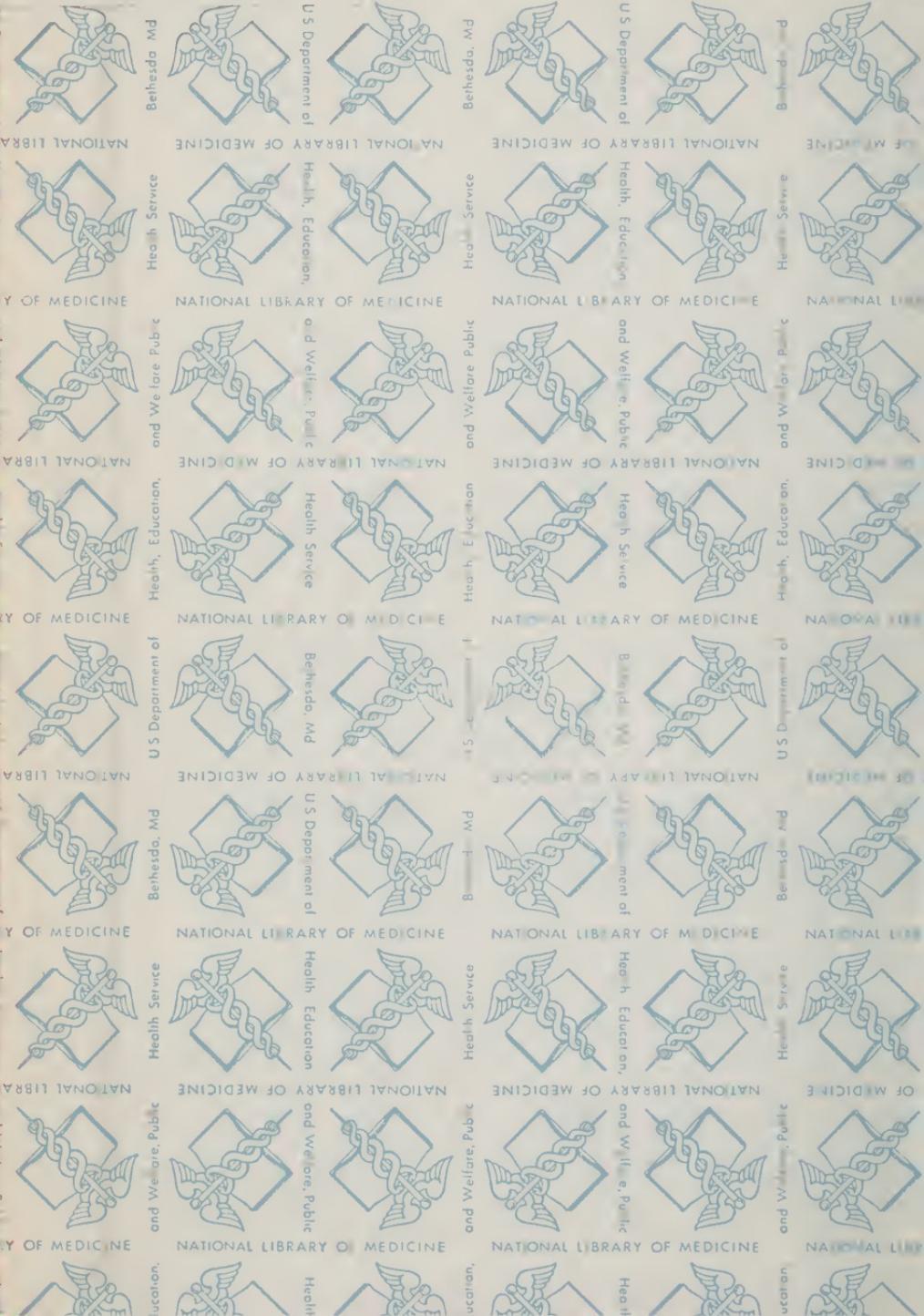


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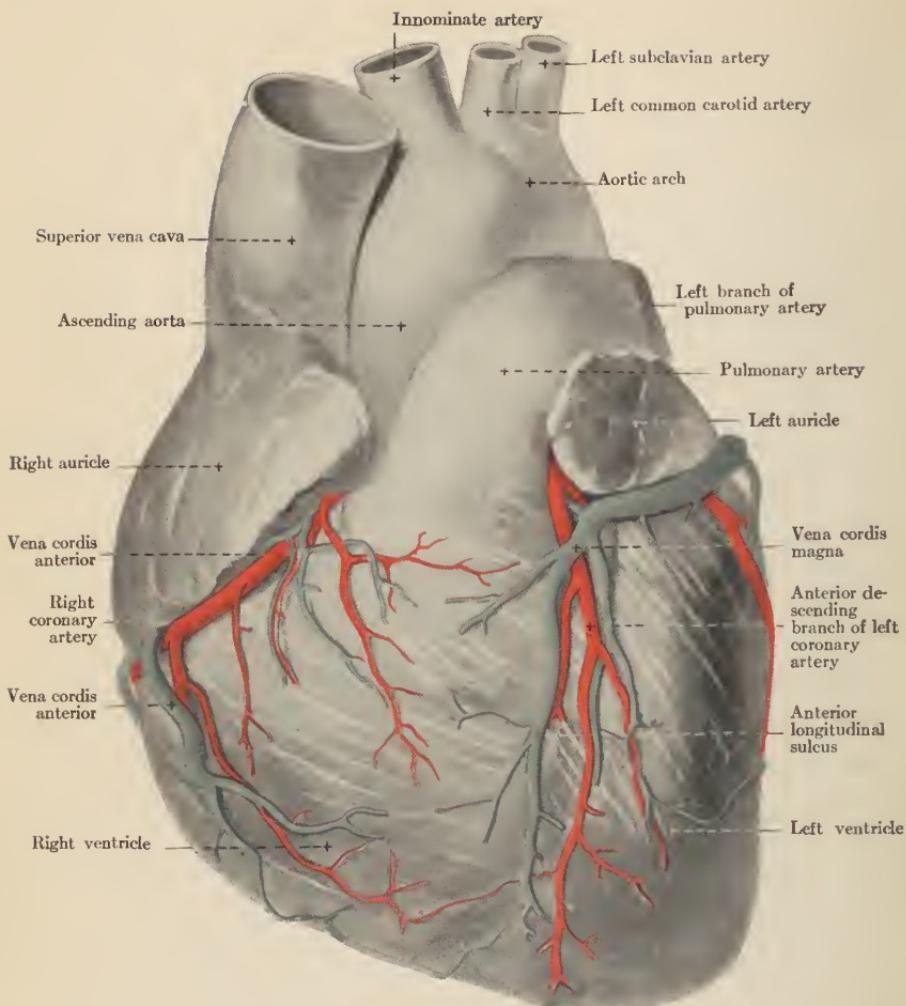


FIG. 1. The heart viewed from above and in front. The surface vessels have been injected.  
(Spalteholz : Handatlas der Anatomie.)

# THE HEART IN MODERN PRACTICE

## DIAGNOSIS AND TREATMENT

BY  
**WILLIAM DUNCAN REID, A.B., M.D.**

CHIEF OF HEART CLINIC AT THE BOSTON DISPENSARY, JUNIOR ASSISTANT VISITING PHYSICIAN AND MEMBER OF THE HEART SERVICE AT THE BOSTON CITY HOSPITAL, FORMERLY ASSISTANT VISITING PHYSICIAN TO OUT-PATIENTS AT THE MASSACHUSETTS GENERAL HOSPITAL.

32 ILLUSTRATIONS



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TO MY FATHER  
ROBERT ALEXANDER REID, M.D.



## PREFACE

IT IS well known that since the introduction of graphic methods of examination, extensive advances have been made in the knowledge of the heart in health and disease. Unfortunately, an acquaintance with this progress in cardiology can be gained only by a rather wide reading of the literature, as, at present, I am aware of no single book in English which is suitable to recommend to the student or physician who asks for the name of a single volume in which he may read up on the subject. The interest in the study of the abnormal rhythms of the heart has been so great that many of the recent authors have limited themselves to a presentation, often admirable it is true, of this aspect alone. There seems, then, in my opinion, to be a real need for a book which will incorporate the best of the new knowledge with that less recently acquired but which may be said to have stood the test of time. Such a book should preferably be brief enough as to be attractive to the bulk of the profession, whose opportunity to read upon a single aspect of medicine is limited. This little book is offered as an attempt, perhaps with indifferent success, to fill the above-mentioned need.

The attention of the reader is invited to the method in which the subject is presented. I refer to the division of the main part of the book into three sections, in which heart disease is described from an etiological, a functional, and a structural viewpoint, respectively. The importance of etiology does not receive sufficient emphasis when it is mentioned only in teaching or in an article contributed to

a medical journal, but if we believe in it, and we do, the text-book should be written in a method that will stamp this on the reader's mind. For instance, is it surprising that the average physician is hazy as to the effects of syphilis on the circulatory organs when it is realized that to find out what is known he must turn to the chapter on valvular affections for an account of aortic regurgitation, to another for syphilitic myocarditis, to another for heart-block and other arrhythmias occurring in syphilis, and to still others for aneurism and angina pectoris? Logically, this material should be brought together in some such manner as I have attempted in the chapter on "*Cardiovascular Syphilis*." The day of the consideration of heart affections from the viewpoint of the structural change or anatomical lesion has passed; of greater importance is that of the functional condition and perhaps even more essential, to an intelligent conception, is the recognition of the etiological type. I have endeavored to present the subject as nearly in conformance with the above belief as is practicable. My excuse for insisting on this point is that it has been made a major feature of this book, which differs thereby from any other book with which I am acquainted.

It may appear that a proper balance has not been maintained between the different subjects. Thus, the chapter on "*Arteriosclerotic Heart Disease*" is of equal length with that on "*Septic Heart Disease*," though the former is of infinitely greater frequency and importance. However, certain subjects lend themselves to a brief presentation, while with others clearness demands greater space. I have sought for brevity with clearness.

Much as it is desirable, it was not found possible to avoid controversial subjects; cardiology is in process of

change and physicians are not in agreement on all points. Whenever a point might be new to some readers, authority has been quoted. It is hoped that the references might stimulate collateral reading.

The electrocardiograms were made by the author in the laboratory of the Boston City Hospital.

It is a pleasure to acknowledge thanks to Austin W. Cheever, who kindly suggested most of the details of the antisyphilitic treatment recommended for cardiovascular syphilis.

W. D. R.



# CONTENTS

## SECTION I

### PRELIMINARY CONSIDERATIONS

CHAPTER	PAGE
I. ANATOMICAL, PHYSIOLOGICAL, AND EMBRYOLOGICAL CONSIDERATIONS .....	15
II. METHODS OF EXAMINATION.....	31
III. METHODS OF EXAMINATION (CONT.)—POLYGRAPHIC EXAMINATION.....	49
IV. METHODS OF EXAMINATION (CONCL.)—ELECTROCARDIOGRAPHIC EX- AMINATION .....	64
V. THE NORMAL HEART .....	84
VI. THE CLASSIFICATION OF HEART DISEASE.....	91

## SECTION II

### TYPES OF HEART DISEASE (ETIOLOGIC)

VII. RHEUMATIC HEART DISEASE .....	97
VIII. SEPTIC HEART DISEASE.....	119
IX. CARDIOVASCULAR SYPHILIS.....	126
X. ARTERIOSCLEROTIC HEART DISEASE.....	142
XI. HYPERTENSIVE HEART DISEASE .....	149
XII. THE HEART IN HYPERTHYROIDISM .....	160
XIII. THE HEART IN DIPHTHERIA .....	165
XIV. CONGENITAL HEART DISEASE .....	170
XV. EFFORT SYNDROME: IRRITABLE HEART .....	175

## SECTION III

### FUNCTIONAL CONDITIONS

XVI. HEART FAILURE—ANGINA PECTORIS .....	183
XVII. THE ARRHYTHMIAS .....	195

## SECTION IV

### STRUCTURAL LESIONS

XVIII. STRUCTURAL LESIONS .....	225
---------------------------------	-----

## SECTION V

XIX. TREATMENT .....	247
----------------------	-----

## APPENDIX

ILLUSTRATIVE CASE REPORTS.....	273
--------------------------------	-----



## LIST OF ILLUSTRATIONS

FIG.	PAGE
1. Anterior view of heart.....	frontispiece
2. Heart with part of interior exposed by the removal of a slice from the anterior surface.....	18
3. Curves of pressure in the left ventricle and right auricle .....	24
4. Comparison between percussion and teleradiography in the determination of the left cardiac border .....	37
5. Arteriograms of pulsus alternans, and of pseudo-alternation .....	52
6. Normal polygram.....	52
7. Sinus arrhythmia .....	52
8, 9. Auricular premature beats. Ventricular premature beat.....	58
10. Paroxysmal tachycardia of auricular origin.....	58
11. Auricular fibrillation.....	59
12. Partial heart-block .....	62
13. Complete heart-block.....	62
14. The Einthoven triangle.....	69
15. Normal electrocardiogram.....	70
16. Ventricular preponderance.....	71
17. Sinus arrhythmia.....	74
18. Auricular premature beats .....	75
19. Ventricular premature beats.....	76
20. Paroxysmal tachycardia.....	77
21. Auricular flutter.....	78
22. Auricular fibrillation.....	79
23. Partial heart-block.....	80
24. Complete auriculo-ventricular heart-block, and intraventricular block....	81
25. Combined arrhythmias.....	83
26. Spirochetes in the myocardium.....	126
27. Syphilitic myocarditis.....	127

## PLATES

PLATE	PAGE
1. Rheumatic heart disease; healed mitral endocarditis with calcification and rupture. Note the thickening and shortening of the chordæ tendineæ.....	98-99 (Courtesy of Timothy Leary)
2. Rheumatic heart disease; acute infectious lesion of branch of the coronary artery with surrounding myocarditis.....	98-99 (Courtesy of Frank B. Mallory)
3. Large vegetations on the mitral valve, in septic heart disease due to the pneumococcus .....	120-121 (Courtesy of Frank B. Mallory)
4. Aneurism of the wall of the left ventricle with thrombus inside, associated with coronary sclerosis and hypertensive heart disease.....	244-245 (Courtesy of Frank B. Mallory)
5. Thrombi in the auricle. Note the corrugated surface of the large thrombus .....	270-271 (Courtesy of Frank B. Mallory)

**SECTION I**  
**PRELIMINARY CONSIDERATIONS**



# THE HEART IN MODERN PRACTICE

## CHAPTER I

### ANATOMICAL, PHYSIOLOGICAL, AND EMBRYOLOGICAL CONSIDERATIONS

THE following salient points of cardiac anatomy, physiology, and embryology have been selected as of most value in the clinical understanding of the heart.

#### ANATOMY

The heart is a somewhat conical-shaped mass of muscle, occupying the pericardial cavity in the middle mediastinum. It is placed obliquely in the thorax; the base is directed backwards and corresponds to the fifth to eighth thoracic vertebræ inclusive, while the apex points downward, forward, and to the left, at a level between the fifth and sixth costal cartilages and a little inside the left mid-clavicular line.

The heart cavity is divided into two lateral halves, and a transverse constriction further divides these latter into two cavities, the posterior on the two sides being called the auricles and the anterior the ventricles. The division of the heart into the four cavities is indicated by grooves upon its surface. In the groove indicating the auriculo-ventricular septum lie the main trunks of the nutrient vessels of the heart.

THE RIGHT AURICLE is composed of a posterior and larger portion, the sinus venosus, and a smaller cavity

placed in front and above, the right auricular appendix. Into the sinus venosus empty the superior and inferior venæ cavæ, and its mesial wall is formed by the interauricular septum.

THE LEFT AURICLE is smaller than the right. It, too, consists of a principal cavity and an auricular appendix. The left auricular appendix is directed forward and to the right so that it overlaps the root of the pulmonary artery. The pulmonary veins, usually four but sometimes three in number, open into the upper part of the posterior wall of the left auricle.

THE VENTRICLES are larger and the muscular layer forming their walls is much thicker than is that of the auricles. *The right ventricle* extends from the right auricle to near the apex and forms the larger part of the anterior surface of the heart. At its base, the right ventricle is prolonged into a conical pouch, the conus arteriosus or infundibulum, from which the pulmonary artery has its origin.

*The left ventricle* is longer than the right, beyond which it projects to form the apex of the heart. It appears on the anterior cardiac surface as a narrow strip along the left border, but forms a considerable portion of the postero-inferior surface. The thickness of the muscular wall of the left ventricle averages about three times that of the right ventricle.

VALVES.—There are four valves in the heart, two between the auricles and ventricles and two between the ventricles and the aortic and pulmonary arteries. These valves are formed by a reduplication of the endocardium and strengthened inside by a fibrous layer. At the attachment of each valve is an annular ring of dense fibrous tissue. The

cusps of the auriculo-ventricular valves are connected at their free margin by the chordæ tendineæ to the papillary muscles. These fibrous bands, or chordæ tendineæ, by limiting the play of the cusps, serve to prevent invagination of the latter into the auricles at the time of ventricular contraction. In the right heart, the valve has three sections or cusps and hence its name, tricuspid. That of the left side has but two cusps and it is termed the bicuspid or mitral valve.

**THE AORTIC and PULMONARY VALVES** each consist of three semilunar segments or cusps, the inner margins of which are free. Near the centre of the free margins of each cusp is a small nodule of fibrous tissue, termed the corpus Aurantii. These latter are to be distinguished from the thickenings produced by endocarditis. At the base of the aorta and of the pulmonary artery are small dilatations opposite each of the semilunar valve segments. These are known as the sinuses of Valsalva.

**THE MYOCARDIUM** consists of bands and layers of muscle tissue intricately interlaced. The fibres of the bundle of His are the only fibres that are common to both auricle and ventricle. MacCallum's studies of the myocardium have disclosed that by dissection the heart may be unrolled so that the four chambers can be separated and weighed separately, a matter of importance in careful studies of ventricular hypertrophy.

**THE SINO-AURICULAR NODE** is a small mass of specialized tissue located at the junction of the superior vena cava with the right auricle and immediately below the endocardium. This node is the seat of origin of the normal heart beat.

THE AURICULO-VENTRICULAR BUNDLE OF HIS (Fig. 2) is the only direct muscle connection between the auricles and ventricles. It arises from near the opening of the

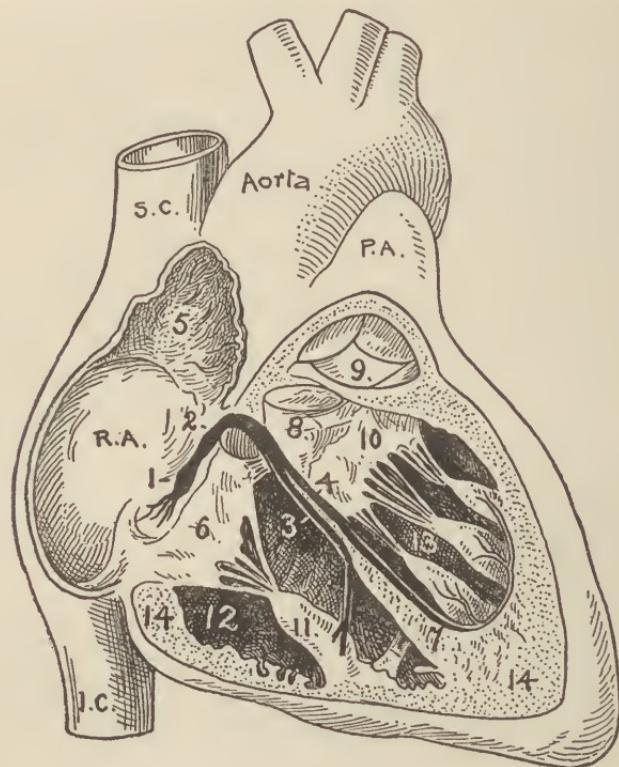


FIG. 2.—Heart with part of interior exposed by the removal of a slice from the anterior surface. The auriculo-ventricular conduction bundle is represented schematically.

S. C.—Superior Cava.	3. Right Branch.	9. Pulmonic Valve.
P. A.—Pulmonary Artery.	4. Left Branch.	10. Mitral Valve.
R. A.—Right Auricle.	5. Right Auricular Appendage.	11. Papillary Muscle.
I. C.—Inferior Cava.	6. Tricuspid Valve.	12. Right Ventricle.
1. Auriculo-ventricular Node.	7. Interventricular Septum.	13. Left Ventricle.
2. Bundle of His.	8. Aortic Valve.	14. Cut Surface of Ventricular Wall.

(*Gray's Anatomy, somewhat modified.*)

coronary sinus, where it is connected with the annular and septal fibres of the right auricle. These fibres converge, form a node (node of Tawara), and continue as a compact

bundle which passes forward to the upper limit of the muscle portion of the ventricular septum, where it divides into right and left branches. These latter course down on either side of the ventricular septum to the right and left ventricles, respectively. Each limb is enclosed in a layer of connective tissue which isolates it from the musculature of the interventricular septum, but in the lower part of the ventricles each branch separates into numerous strands which enter the papillary muscles and spread over the entire inner surface of the ventricular muscle and form histological connections with the true cardiac muscle fibres. The right limb is the smaller of the two and usually enters the anterior papillary muscle by passing along the moderator band when that is present. The bundle of His consists of narrow, somewhat fusiform fibres, but the two branches and their terminal strands are composed of Purkinje fibres.

A bursa or lubricating mechanism is in relation with the main bundle, according to Curran,<sup>1</sup> and a special artery arising from the right coronary enters and follows the bundle. These facts suggest the possibility of the occurrence of a bursitis from acute articular rheumatism or other infections, as causing certain cardiac symptoms.

The PURKINJE FIBRES are very much larger than the cardiac cells, and differ from them in several ways. In longitudinal section they are quadrilateral in shape. The central portion of each fibre contains one or more nuclei and is made up of granular protoplasm, with no indication of striations, while the peripheral portion is clear and has distinct cross striations. The fibres are intimately connected with each other, possess no definite sarcolemma, and do not branch.

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<sup>1</sup> *The Anatomical Record*, 1909, vol. iii, No. 12: 618.

The ARTERIES supplying the heart are the right and left coronaries. The right coronary artery rises from the anterior aortic sinus of Valsalva, passes forward to the auriculo-ventricular groove and after coursing to the right, divides into a transverse branch, which continues in the auriculo-ventricular groove to anastomose with the transverse branch of the left coronary artery, and a descending portion which supplies the right and posterior surface of the heart and finally anastomoses with the descending branches of the left coronary at the apex.

The left coronary has its origin in the left posterior sinus of Valsalva of the aorta. It, too, divides into a transverse and a descending branch. The former passes to the left in the auriculo-ventricular groove to anastomose with its fellow from the right coronary, and the descending branch, after supplying the left and part of the anterior cardiac surface, descends to the anastomosis at the apex.

These vessels occasionally arise by a common trunk or their number may be increased to three or more.

VEINS.—The coronary sinus receives the majority of the veins draining the blood from the substance of the heart. It terminates in the right auricle between the opening for the inferior vena cava and the tricuspid valve. Its orifice is guarded by a semilunar valve (the coronary valve).

The anterior cardiac veins, comprising three or four small vessels, empty direct into the right auricle. The veins of Thebesius, consisting of a number of minute vessels arising in the substance of the heart, empty mostly into the auricles, but a few empty into the ventricles.

The NERVES of the heart are derived from the superficial and the deep cardiac plexuses, and from these plex-

uses obtain fibres from the vagus and sympathetic nerves. The superficial cardiac plexus lies under the arch of the aorta, while the deep cardiac plexus is under the tracheal bifurcation. The nerves from the plexus are freely distributed on the surface and in the substance of the heart, many of the separate filaments being furnished with small ganglia.

The PERICARDIUM is a serofibrous sac in which the heart and the commencement of the great vessels are contained. It consists of an outer fibrous layer and an inner layer of serous type. The fibrous layer blends with the central portion of the diaphragm and with the external coat of the great vessels. There are several prolongations of this outer layer (called ligaments of the pericardium) which attach it to the sternum, diaphragm, and vertebral column (by way of the cervical fascia). On the lateral surface of the pericardium is found the phrenic nerve as it passes downward to the diaphragm. The serous layer lines the fibrous pericardium and is invaginated by the heart so that it is described as consisting of a parietal and of a visceral layer.

The INTERNAL MAMMARY ARTERIES, to be avoided in paracentesis of the pericardium, descend on the inner thoracic wall about one half an inch from the sternal margins.

The PULMONARY ARTERY rises from the conus arteriosus slightly anterior and to the left of the aorta. It passes obliquely to the left and under the arch of the aorta, where it divides into right and left branches. The artery averages but two inches in length. It is connected to the aortic arch by a short fibrous cord, the ligamentum arteriosum. This latter is the remnant of the fetal ductus arteriosus.

The AORTA rises from the base of the left ventricle, ascends a short distance, then arches backward and to the left to descend on the left side of the vertebral column. The ascending portion starts behind the left half of the sternum at the level of the lower border of the third costal cartilage and passes upward and forward to the right to the level of the upper border of the second right costal cartilage. The ascending portion is contained within the pericardium. When the aorta is distended it is about one quarter of an inch from the posterior surface of the sternum. This space is occupied by loose areolar tissue and the remains of the thymus gland.

The transverse arch of the aorta lies upon the trachea, esophagus, and thoracic duct, and the left recurrent laryngeal nerve winds around it.

## PHYSIOLOGY

THE EVENTS THAT OCCUR DURING A SINGLE CARDIAC CYCLE.—A complete cardiac cycle is the time from any given feature of the heart beat until that feature is again produced. For example, if we begin with the closure of the semilunar valves, the sequence of events is as follows:—At that moment the second heart sound is heard and the ventricle is quickly relaxing from its previous contraction. For a brief interval the auriculo-ventricular valves remain closed and for the moment the ventricles are shut off on both sides. The blood is flowing steadily into the auricles and dilating them. As soon as the ventricles relax completely the pressure of the blood in the auricles presses open the auriculo-ventricular valves, and from that moment until the beginning of auricular systole the blood from the large veins is filling both auricles and ventricles.

The ventricular walls become more tense and the auriculo-ventricular valves are floated into position ready for closure. The auricular systole sends a sudden wave of blood into the ventricles, which closes the auriculo-ventricular valves.<sup>2</sup> The contraction of the auricles also momentarily blocks or retards the flow from the large veins, whence the auricular wave in the jugular veins. Ventricular systole follows immediately upon auricular systole. As the ventricles enter into contraction, the first sound is audible, and for a brief moment the ventricular cavities are again shut off on both sides. Soon the rising intraventricular pressure opens the semilunar valves and a stream of blood is forced into the aorta and pulmonary artery. During ventricular systole, the auricles continue to receive blood from the large veins; the venous flow is never completely blocked, but at most experiences a slight retardation during the very brief auricular systole. At the end of ventricular systole the intraventricular pressure falls below that in the aorta and pulmonary artery and the excess of arterial pressure closes the semilunar valves, thus completing the cardiac cycle.

Study of the diagram, Fig. 3, showing the relation of the pressure in the auricle, ventricle, and aorta, should aid one to visualize the events in a cardiac cycle. The latter is commonly divided into two periods: that of contraction or systole, and that of relaxation or diastole. At an average heart rate of 72 per minute, the cardiac cycle occupies about 0.8 of a second, and this may be divided approximately as follows: auricular systole 0.1, ventricular systole 0.3, and the common pause of the heart as 0.4 of a second, respectively. Diastole, as commonly determined, is the

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<sup>2</sup> HENDERSON, Y., and JOHNSON, F. E.: *Heart*, 4: 69, 1912-1913.

period of ventricular diastole, and auricle systole is described as late diastolic or presystolic. In hearts beating at a slow rhythm, the greater part of ventricular filling may be completed in early diastole, which is then followed by

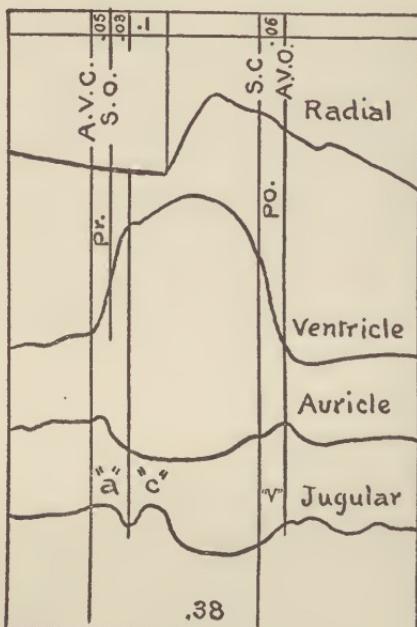


FIG. 3.—Curves of pressure in the left ventricle and right auricle; the radial and jugular pulse tracings are placed above and below to show their time-relations.

A. V. C. and A. V. O., closure and opening of the auriculo-ventricular valves, respectively; S. O. and S. C., opening and closure of semilunar valves, respectively; pr., presphygmic period; po., post-sphygmic period. The time intervals of some of the periods are marked above and below in seconds.

(This diagram is from one published by Thomas Lewis, with slight modification.)

a period of comparative rest. This resting phase is termed diastasis.

Contrary to the general impression, the apex of the heart, from which the pericardium has not been removed, does not twist forward and to the right with contraction, but, according to Starling,<sup>3</sup> there is then a definite descent

<sup>3</sup> STARLING, E. H.: Human Physiology, third edition, 1920, p. 947.

of the base downward toward the apex. The heart tends to be fixed by its attachment to the venæ cavæ. This fact is a factor in an enlargement of the right ventricle being manifest to the left rather than to the right.

The HEART SOUNDS.—The first heart sound is largely muscular in origin. It begins with the “setting” of the ventricles, and continues until the highest intraventricular pressure has been produced. The systole of the ventricles then continues in silence the discharge of blood into the aorta until its sudden termination, which is marked by the second sound. The second sound is associated with the closure of the aortic and pulmonary valves. The actual sound is due to the relaxed semilunar cusps suddenly being put into a state of tension; a comparable sound may be produced by suddenly drawing taut a fold of a handkerchief held between one's hands. A second sound is possible without a perfect closure of the valve. The closure of the mitral and tricuspid valves is relatively quiet and probably contributes but little to the volume of the first heart sound.<sup>4</sup> The loudness and intensity of the first sound has been found<sup>5</sup> to be directly proportional to the intracardiac tension, and especially to the tension developed during the isometric period *i.e.*, before the opening of the semilunar valves.

MECHANISM OF THE HEART BEAT.—On inspection the exposed heart is seen to contract and relax in rhythmic succession. The contraction wave starts at rhythmic intervals at the sino-auricular node, then spreads centrifugally over the auricles, like the rings from a stone thrown into a millpond, meets a slight delay at the auriculo-ven-

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<sup>4</sup> REID, W. D.: *Jour. Amer. Med. Assn.*, 76: 432 (Feb. 12, 1921).

<sup>5</sup> WIGGERS, C. J.: *Arch. Int. Med.*, 24: 471 (Nov., 1919).

tricular bundle, and thence proceeds through the right and left branches to the Purkinje fibres, where it is distributed to the muscular fibres of the ventricular walls. Other parts of the specialized tissue, namely, the auriculo-ventricular (Tawara) node and the conduction bundle on the ventricular side of the node, have the power of stimulus production, but the rate of this is slower than that of the sino-auricular node, and therefore, save in abnormal conditions, the heart contracts in response to stimuli received from the higher centre, the sino-auricular node.

It now seems established that the myogenic theory of the production and transmission of the heart beat is correct. The nerves have been shown to control the heart beat but do not initiate it or conduct the contraction wave. The vagus inhibits and the sympathetic accelerates the beat, each counteracting the other. The heart with its nerve connections severed may continue to beat.

Immediately after the muscle fibres of the heart have contracted they exhibit a refractory stage in that they fail to respond to stimulation. Restoration of excitability at once commences and increases progressively during diastole. When the cardiac muscle is stimulated it either does not contract at all, or does so to the fullest possible extent at the time. The amount of contraction does not depend on the strength of the stimulus employed, but varies according to the time at which the stimulus is received. When a stimulus is received early in diastole the resulting contraction is weaker than that of the previous cardiac cycle. Within limits, the amount of contraction varies directly in proportion to the length of the previous diastole. This principle serves to explain the fact that the more

rapid the heart rate the greater is the possibility of cardiac failure.

Gaskell has described five fundamental functions of cardiac muscle, namely: stimulus production, excitability, conductivity, contractility, and tonicity. The terms are somewhat theoretical, to be sure, but very useful in the discussion of the various features of the heart's activity. In abnormal conditions of the heart it is rare that the disturbance is limited to but one of the five fundamental functions mentioned above.

**THE PHYSIOLOGY OF MUSCULAR EXERCISE.**—The output of the heart varies from 3 to 5 litres per minute in individuals at rest, it is raised by moderate exercise to 8 or 9 litres, and during heavy muscular exercise it may reach 20 litres or even more. It is of importance to consider briefly the conditions associated with this increase in the cardiac output.

At the onset of exercise, a constriction of the splanchnic vessels takes place and this diverts much of the blood into the muscles, and increases the pressure in their vessels. The increase in the blood supplied to the active muscles enables the latter, by their contractions, to expel a greater volume of blood into the veins and thence back to the heart. The heart then drives the blood out again into the muscles. Thus, in a certain sense, it can be seen that the mechanism, by which during exercise a greater volume of blood circulates through the active muscles, is largely dependent upon the activity of the latter. If the exercise is continued, some dilatation of the vessels in the working muscles usually occurs, and this results in a further increase in the blood supplied to these same muscles and enables them to return more blood to the heart.

It has been said above that during exercise there is a greater volume of blood returned to the heart, which is then called upon to propel it forward if the increased circulation is to be maintained. In other words, the output of blood from the heart must be augmented. The physiologists state that this may be accomplished by an increase in the number of contractions per minute and by a greater output per beat. The former certainly occurs, but as regards the latter there is some disagreement.

During diastole the heart is filled passively by the blood flowing in from the veins, and if the pressure of the latter is increased (in exercise) the heart undergoes a greater distention or dilatation. Dilatation is, therefore, a perfectly normal process, and the extent to which it can take place, under physiological conditions, is ultimately limited by the pericardium.<sup>6</sup>

It has been shown<sup>7</sup> that the ventricles never pass in a single systole from utmost fulness to extreme contraction. The same observers hold that under normal conditions (*i.e.*, when the pressure of the venous return is not decreased) the amplitude of the beat is determined by the duration of diastole; in other words, the systolic discharge is less when the heart rate is accelerated. The output per beat may be increased, however, during exercise when the heart rate is not quickened, as apparently occurs in individuals habituated to exercise.

An understanding of the physiology of exercise makes more intelligible many of the phenomena observed in the study of the heart both in health and in disease. The

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<sup>6</sup> BAINBRIDGE, F. A.: *The Physiology of Muscular Exercise*, Longmans, Green and Co., 1919, p. 54.

<sup>7</sup> HENDERSON, Y., and BARRINGER, T. B., Jr.: *Amer. Jour. Physiol.*, xxx: 366 (March, 1913).

subject is, unfortunately, complex and further presentation will not be attempted here.

## EMBRYOLOGY

The CIRCULATORY SYSTEM in the early embryo is that of a single tube or artery whose wall at a certain point possesses the power of rhythmic contractions. The specialized part of this primitive tube later undergoes a sort of S-shaped twist, and by a complex process of evolution develops into the heart. There are many modifications which may occur in the above-mentioned development, and some of these are recognized in the condition termed "congenital heart." The variability in the relation of the right and left vagus nerves to the sino-auricular node and the auriculo-ventricular bundle may, in part, be due to differences in the evolution of the heart. There are several structures in the heart which possess particular significance as regards the fetal circulation.

The FORAMEN OVALE is in the interauricular septum and is freely open until about the middle of fetal life, when a fold of tissue grows up from the posterior wall of the left auricle and acts as a valve in permitting the passage of blood only in the direction of right to left auricle.

The EUSTACHIAN VALVE is a fold of tissue projecting upward in front of the orifice of the inferior vena cava and tends to divert the blood from this vessel through the foramen ovale into the left auricle.

The DUCTUS ARTERIOSUS (ductus Botalli) is a short tube, about 10 mm. in length at birth and 2 mm. in diameter. In fetal life it forms the continuation of the pulmonary artery, and opens into the aorta just beyond the origin of

the left subclavian artery. It conducts the greater part of the right ventricular blood stream into the aorta.

The upper and posterior part of the INTERVENTRICULAR SEPTUM, which separates the aortic vestibule from the lower part of the right auricle and the upper part of the right ventricle, is derived from the inferior part of the aortic septum of the fetus. Owing to defective development of this septum, an abnormal communication may exist at this spot.

**CHANGES IN THE CIRCULATION AT BIRTH.**—Soon after birth the development of the circulation in the lungs increases the pressure in the left auricle and the effect of this is to press the valve-like fold against the ring of the foramen ovale. The structures fuse and closure of the foramen is usually complete by about the tenth day. A further result of the onset of respiration is the diversion of the blood through the pulmonary arteries. The ductus arteriosus begins to contract and is usually completely closed by the fourth to the tenth day. Occasionally a small lumen persists.

The changes in the circulation elsewhere in the body need not be described in this brief résumé.

## CHAPTER II

### METHODS OF EXAMINATION

CARDIAC diagnosis is built up from the data collected by a variety of methods. Care and thoroughness are just as important in the diagnosis of heart as in other medical conditions. The physician who limits his examination to auscultation displays a lack of appreciation of the subject.

**HISTORY.**—A careful general history should be obtained as a preliminary to one bearing more directly on the heart. The presence or absence of diseases known to cause heart affections is to be determined. Of special etiological importance are various infections, such as rheumatic fever, chorea, tonsillitis, scarlet fever, pneumonia, puerperal sepsis, etc. If present, questions should be directed to develop the genuineness and severity of the above. A history of syphilis is always important. If the patient is said to have been treated in the past for cardiac disease, the details of this should be explored.

The anamnesis should contain data as to the tolerance of exercise. A knowledge of the nature of the patient's occupation, and whether it is pursued without symptoms, is often most useful. A statement as to the effect of climbing stairs and walking on the street is helpful in estimating the tolerance to physical exercise. In children, one may inquire if the patient can run and play games as well as other children.

The nature and duration of symptoms, if any, should be noted. The more common of these are shortness of breath, cough, palpitation, pain, indigestion, undue fa-

tigue, etc. Sufficient details are indicated of any symptoms that may be present. Thus, it is not enough to note that the symptom of palpitation is present, for intelligent questioning may at times produce a description of attacks of paroxysmal tachycardia. So-called nervous symptoms should be duly recognized.

**INSPECTION.**—The patient may present the mitral facies. Dyspnoea and cyanosis suggest some degree of heart failure. Visible pulsation of the carotid arteries, which is sometimes accompanied by a slight nodding or shaking of the head, points strongly to insufficiency of the aortic valve. Distention and visible pulsation of the external jugular veins is associated with engorgement of the right auricle. Exophthalmos and other signs of Graves's disease should bring to the examiner's mind the possibility of the heart in hyperthyroidism.

Inspection should take note of the position and character of the apex impulse, if present. Any abnormal pulsation or retraction requires explanation; depending on position and character, they may point to an aneurism, enlargement of the right ventricle, adhesive pericarditis, etc. Bulging of the precordia is common in heart disease beginning in childhood. Clubbing of the fingertips may be associated with congenital heart disease and severe heart affections in early life. Epigastric pulsations may be due to an enlarged right heart, but as they occur in low-lying hearts, and rarely even in marked left ventricular hypertrophy, the sign must be interpreted with caution.

The legs may be obviously swollen from edema, and in the more marked cases the swelling may extend to the genital region and trunk. As contrasted with edema of renal causation, that due to cardiac failure is more directly

influenced by gravity. It should be remembered, however, that serious heart failure may be present without edema. The true reason for edema is obscure; that it is due solely to circulatory stasis, no longer seems a satisfactory explanation. Some chemical process has been suggested.

**PALPATION.**—The situation and extent of the apex beat, or point of maximum impulse, should be determined. In cases in which the apex beat is not palpable, it may become so if the patient is directed to lean forward to the left and to exhale fully. It is generally best to palpate with the tips of the fingers starting outside of the left border of the heart and proceeding inward. If the impulse is diffuse, the point of maximum forward thrust is to be taken as the apex beat. Its location is to be recorded laterally in relation to the midclavicular line (nipple line is about as accurate in male subjects) or other chest line, and vertically by the number of the thoracic interspace.

It is suggested that in determining the number of the interspace, advantage be taken of the anatomical fact that the transverse ridge marking the junction between the manubrium and gladiolus of the sternum (often termed angle of Ludwig) is directly opposite the sternal articulation of the second rib. Starting with a finger placed above and another below this rib, by progressive straddling of the ribs downward, one can accurately determine the interspace in which the apex beat presents.

The position of the patient should be considered in drawing any conclusions from the location of the apex impulse. Thus, in the dorsal decubitus, the beat commonly moves up one interspace from that of the sitting or standing position. The manœuvre of palpating the impulse when the patient is lying on the left side is useful

to bring out the mobility of the apex. An unduly wide swing to near the anterior axillary line or even further is often disclosed in hypertrophy of the left ventricle, but in this case the impulse in the axilla is really due to the lateral thrust of the wall and not of the apex of the enlarged ventricle. In some cases, where pericardial or pleuro-pericardial adhesions are present, the apex beat may remain fixed. An abnormal position of the apex impulse may be produced by pleural effusions, ptosis of heart, and congenital or acquired dextrocardia. In insufficiency of the aortic valve, the apex impulse tends to be displaced downward and to the left, whereas in mitral stenosis the displacement, if present, is horizontal and to the left.

The character of the cardiac impulse, best observed by palpation with the flat of the hand, may give further data as to the condition of the heart beneath. Thus if there is hypertrophy of the left ventricle, as in aortic regurgitation, the impulse feels heaving and sustained, while in many cases of mitral stenosis, the sensation is as though the chest wall were hit a sharp blow or tap which was as quickly withdrawn.

The precordia should be palpated for the detection of any other pulsations and thrills. The loud purring thrill should be distinguished from the slight vibration, or "suggestion of a thrill," which may be found in merely irritable hearts.

It is often necessary to combine auscultation with palpation in order to properly time pulsation and thrills. Thus an impulse over the body of the right ventricle may be found to be diastolic in time, with a systolic recession, and is good evidence of right ventricular enlargement. When this sign is present, it is the rule to find the apex

impulse absent or poorly marked. Palpation of the carotid pulsation in the neck is a reliable method of checking the time of the heart sounds.

The hand placed over the second left costal cartilage may detect a forcible closure of the pulmonic valve. In mitral stenosis and in gallop rhythm there may be a double diastolic shock of the apex. Friction fremitus may be palpable in some cases of pericarditis. In interpreting an impulse near the head of the sternum as due to an aneurism or a dilated aorta, it should be remembered that when a deformity of the thorax and spine are present, a normal aorta may cause such an impulse, due to its abnormal position in close contact with the chest wall. Pulsation of the first part of the aorta may be palpated in the suprasternal notch in case the aorta is elongated or dilated as in arteriosclerosis or aortitis, and also in patients in whom the vessels are merely atonic, or in whom the diaphragm is unduly high.

**PERCUSSION.**—The development of radiography has disclosed the limitations of percussion in determining the size and shape of the heart. In fact, some authorities go to the extreme of holding that percussion is valueless as a means of diagnosing the presence or absence of cardiac enlargement. With this opinion the writer is not in agreement. But it is desirable that the limitations of information obtained by means of percussion alone, should be more widely appreciated. A valuable contribution to this subject was made in 1916, by G. C. Shattuck and others, at the Massachusetts General Hospital.<sup>1</sup>

Percussion that is of value is an art; it cannot be learned

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<sup>1</sup> SHATTUCK, G. C.: *Boston Med. and Surg. Jour.*, clxxiv: 301 (March 2, 1916), see also *Internat. Clinics*, iv, series 29: 106, 1919.

from books, but is acquired by experience from the clinical examination of patients, and its margin of error will be lessened if frequently checked by postmortem findings and especially by X-ray mensuration of the heart and aorta. It should be understood that skilled examiners may differ in the demarcation of a particular cardiac border, but that each may, as a result of experience in practicing percussion according to his own method, correctly determine the presence or absence of enlargement of the heart. Since there is no normal-sized heart (the heart fits the patient and the shape of the thorax is subject to much variation) the measurements of the borders of cardiac dulness should always be recorded in relation to the chest lines. Thus the written statement that the left border of cardiac dulness extends to "10 cm. from the midsternal line" becomes informing to the reader, who may not have the patient available for personal examination, if there is added the relation to the midclavicular (nipple line is accurate enough in the male sex) or axillary lines, as "1 cm. outside the midclavicular line," etc. The latter statement is more valuable than the former.

Due allowance must be made for the difference in cardiac shape and location that is dependent upon the position of the patient. Thus the contour of the heart broadens and the organ assumes a higher level in the thorax when the patient changes from the standing or sitting to the horizontal posture. Still another factor that adds to the difficulty of accurately determining the size of the heart by percussion, is that the organ is subject to a rhythmic diminution and increase in magnitude coincident with systole and diastole. This latter difficulty is met, in part at least, by repeating the percussion stroke and

being guided by the average result. Both light and medium percussion are indicated, and the border of cardiac dulness is generally best appreciated if the percussion proceeds from over the lung resonance toward the heart, rather than the reverse way.

It should be appreciated that when the border of cardiac dulness extends around the curve of the chest wall, it is impossible for the percussion finding to agree exactly

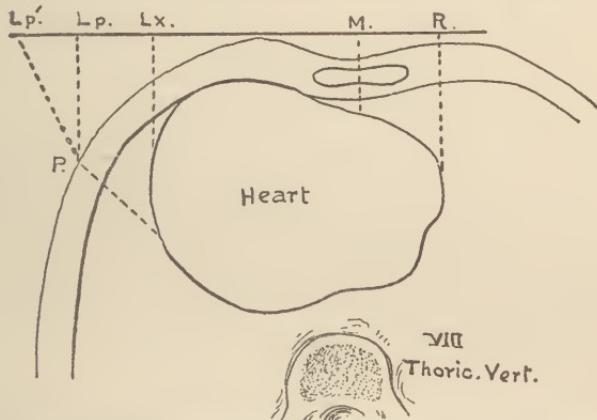


FIG. 4.—Comparison between percussion and teleradiography in the determination of the left cardiac border. R, right cardiac border; M, midsternal line; Lx, Lp, and Lp', the left cardiac border as determined by the X-ray, percussion with the measuring tape held horizontal, and when the latter is applied around the curve of the chest, respectively. P, point on chest wall where left border of cardiac dullness begins. Diagram based upon a frozen section.

with the measurement obtained by the Röntgen examination. The discrepancy is due to a simple problem of physics which will be evident from a glance at the accompanying diagram. (Fig. 4.)

The right border of cardiac dulness is notoriously difficult to obtain. It demarcates the extension to the right of the right auricle, which, it should be remembered, lies at a level distinctly posterior to the chest wall. Also its proximity to the sternum introduces the factor of conduction of the sound along that bone.

To determine the curve of the left border of the deep cardiac dulness is often of value in physical examination. Normally it is convex inwards; a straightening or outward bowing of this line should suggest enlargement of the left auricle and the infundibulum, *i.e.*, the part of the right ventricle adjacent to the origin of the pulmonary artery. The radiographer often reports somewhat as follows, "The heart is enlarged laterally, and is especially prominent in the region of the auricles. The picture is that of mitral stenosis." This prominence of the left auricle can often be detected by percussion. The left auricle, of course, lies at a distinctly posterior level, and it is evident that an increase in the heart dulness to the left, at the level of the third and second intercostal spaces, is in part due to enlargement of the infundibulum.

An increase in the area of absolute heart dulness may be found in enlargement of the right ventricle.

Percussion outside the nipple, with the patient lying and turned towards the left side, will often bring out enlargement backward of the left ventricle. C. F. Hoover<sup>2</sup> urges that percussion be performed with the patient sitting up and leaning forward, as then the heart is in better contact with the chest wall.

Examination by percussion should always include the region over the great vessels. The writer has found the bent-finger method of Plesch<sup>3</sup> of assistance in delimiting the extension to right and left of the supracardiac dulness, in enlargement of the first part of the aorta.

In the presence of pericarditis with effusion, percussion

<sup>2</sup> HOOVER, C. F.: *Jour. Amer. Med. Assn.*, 75, 24:1626 (Dec. 11, 1920).

<sup>3</sup> BRUGSCH and SCHITTENHELM: *Lehrbuch klinischer Untersuchungsmethoden*, Urban and Schwarzenberg, Berlin and Vienna, 1911, p. 57.

may disclose the heart dulness to be pear-shaped, with the small end towards the base of the heart. In large effusions, there may be an obliteration of the angle between the percussion line of the right auricle and the upper border of the liver.

A knowledge of the anatomy of the heart and of the various shapes which the latter may assume, and an appreciation of the limitations of percussion, are most important in making valuable this method of examination.

**AUSCULTATION.**—Success in auscultation of the heart requires the use of a stethoscope equipped with both the diaphragm type of chest piece, such as is found in the Bowle's stethoscope, and some sort of bell attachment. Most observers, and rightly so, acquire most of their skill with either the diaphragm or the bell-shaped chest piece, but they should be familiar with both, since experience has shown that there are murmurs which are better appreciated and at times only detected by one or the other attachment. Thus, the relatively high-pitched, diastolic murmur of aortic regurgitation is best heard when using the large diaphragm chest piece, while it often happens that it is necessary to change to the bell to detect the low-pitched apical diastolic murmurs of mitral stenosis.

The late Austin Flint offered a reasonable explanation of the above in the following: "All elastic structures, however, have their own vibration times, and when oscillations are transmitted to them at that rate they are set in motion. This is daily seen in the laboratory, when, as an ill-balanced centrifuge is gradually increased in speed, now one, and now another pipette, rack, beaker, or burette begins to rattle, and subsides only when the centrifuge's vibrations have altered to become more rapid. It is this which makes it necessary that marching troops break step while crossing

bridges, and upon this same fundamental law depends the fact that, if the sound waves of one tuning fork reach a tuning fork, string, bell, or other sonorous body of similar pitch, the latter is set in action. This law is made of practical use in analyzing sounds composed of several tones, by exposing a series of resonators to the compound vibration and noting which resonators are set in covibration. It is also of great importance in the construction of stethoscope bells. When dealing with faint murmurs of low pitch, the old Gannet type of stethoscope not infrequently settled an argument, because the large resonating cavity of its bell intensified murmurs of that pitch."<sup>4</sup>

Direct auscultation is performed by applying the ear of the observer direct to the patient's chest. For esthetic reasons a thin towel or handkerchief may be employed to prevent actual contact with the patient's skin. This method of auscultation is especially useful in the detection of aortic murmurs and is most frequently resorted to by those who do not use the large diaphragm attachment to their stethoscope.

After noting the general character of the sounds, the rhythm, etc., it is commonly necessary that the auscultator should concentrate his attention on individual sounds or murmurs, excluding for the moment the other auscultatory phenomena (a trick that can readily be learned). It is essential to time the sounds in relation to the heart cycle. In most instances this may be judged correctly by the character of the sounds, but it is a good rule to always obtain confirmation by inspection or palpation of the apex or carotid pulsation. To the beginner, it is suggested that he consciously direct his attention to noting the intensity,

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<sup>4</sup> A Manual of Physical Diagnosis, by Austin Flint, eighth edition, revised by H. C. Thatcher. Lea & Febiger, 1920, p. 22.

duration, point of maximum intensity, range of transmission, etc., of each sound or murmur. With experience, much of this becomes subconscious.

No auscultation may be said to be complete unless the patient is examined both while in the sitting or standing position and also in the horizontal posture. The position of the patient influences the contact of the heart with the chest wall and therefore has a direct bearing on the transmission of the various murmurs. In the main, basal murmurs are of maximum audibility when the patient inclines his chest well forward, while the apical bruits are better appreciated when the patient is lying on the back or lying turned obliquely toward the left. Further emphasis will be laid on these points in the discussion of the various valve lesions.

The effect of exercise on the auscultatory phenomena should always be noted. An acceleration of the heart rate, as a result of exercise, may cause an irregular rhythm to be displaced by one that is regular, and serves to rule out auricular fibrillation. Exercise, by increasing the velocity of the flow of the blood through the heart, may enable the examiner to hear certain murmurs, or may so increase the audibility of some suspicious sound, that a decision can be made with confidence as to whether it is a murmur or not.

The inhalation of the fumes of a pearl of amyl nitrate appears to act very similarly to exercise in its effect on the heart sounds and murmurs. To the writer it appears to offer no particular advantage.

Accuracy in auscultation, of course, requires experience. At least average acuteness of hearing is necessary. It would seem that the two greatest contributors to a suc-

cessful result are, first, the ability to analyze the findings present by the concentrating of the examiner's attention on the sounds heard with one phase of the heart cycle at a time; and, second, the deliberate directing of his mind toward the detection of the auscultatory phenomena, whose possible presence is suggested by a clear-cut knowledge of heart affections.

**BLOOD PRESSURE DETERMINATION.**—This is usually accomplished by the auscultatory method, using a pressure cuff over the brachial artery and placing the end of the stethoscope over the vessel, just distal to the bandage wrapped around the arm. Sufficient instructions are furnished by the instrument makers. The aneroid type is nearly as accurate as that in which a column of mercury is employed, and for all clinical purposes either type is suitable.

The sounds heard in the auscultatory method, when the pressure in the cuff is allowed to fall gradually, may be divided into five phases: (1) the sudden appearance of a clear sound, (2) acquisition of a murmurish quality, (3) replacement of the murmur by a sound which becomes progressively louder, (4) muffling of the sound, (5) disappearance of the sound. The first phase is generally taken as the systolic pressure and the fifth as the diastolic level, the difference between them being the pulse pressure. When the Corrigan type of pulse is present, sometimes all sound may not disappear. In that case it is necessary to use the fourth phase, which is estimated to be about 5 to 6 mm. higher.

In the palpitory method, the arm band is applied as above and the reading is taken the moment the finger detects the return of the pulse in the radial artery as the

pressure in the cuff is allowed to fall. In this method the reading averages 5 to 10 mm. of mercury lower than in the auscultatory method. The diastolic pressure cannot be obtained by palpation. Both in health and in association with aortic regurgitation, the systolic may be higher and the diastolic lower in the femoral than in the brachial artery. Starling<sup>5</sup> explains this as due to the summation of waves reflected from the periphery. The femoral artery is larger, its walls are more rigid and the peripheral resistance is more localized; all of these contribute to make the reflected waves greater.

The blood pressure is much influenced by psychic and mechanical conditions. A single reading is of but limited value, as there is too great a chance that the result may not represent the usual or average pressure level in the patient under examination. The test should be repeated on several occasions under as nearly the same conditions as possible. Care should be taken to avoid exposure to cold, and the physical and mental relaxation of the patient should be sought. Except under the most carefully controlled conditions and when readings are taken over a period of twenty minutes or more, the figures should be regarded as mere approximations. The hemodynamics of blood pressure are too subtle to be considered in an off-hand manner.

**EXERCISE TOLERANCE.**—An estimate of the response to exercise or, as it is commonly termed, the exercise tolerance, is essential in forming an accurate opinion of the heart which is under examination. Many procedures have been proposed in the endeavor to obtain a scientific estimate of the cardiac efficiency, but since, in the author's opinion,

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<sup>5</sup> STARLING, E. H.: Principles of Human Physiology, 3rd edit., 1920. Lea and Febiger, p. 971.

their value is doubtful or as yet unsettled, they will not be described.<sup>6</sup>

It is not rare to find individuals who can satisfactorily perform some set test, such as lifting a pair of dumbbells fifty times, but yet cannot perform their daily tasks without circulatory symptoms. Nervous factors frequently interfere with the reliability of tests carried out in the physician's office. Of course, since patients differ so widely in age, weight, stature, sex, and habituation to physical effort, it is impracticable to use the same exercise for all. It seems sounder to subject each case to some physical effort suitable to the individual (an obese, middle-aged woman should hardly be asked to bend forward until the finger tips touch the floor, while the knees remain unflexed) and which will cause a definite elevation of the heart rate. And then one should carefully watch for effects such as breathlessness, cyanosis, undue elevation of the heart rate, etc., which are untoward for the individual under examination. It follows, then, from the above statements, that the estimation of the exercise tolerance remains a matter of the physician's judgment and not a clinical fact like the blood count. Any attempt to resort to a numerical basis appears to the writer to be fallacious unless controlled by a clear knowledge of its limitations and its physiological background. It should be remembered that exercise, if pushed far enough in a person with a normal heart, will cause symptoms and signs similar to those produced in a patient with some degree of cardiac failure. Experience is the best guide in judging when the effects of exercise may be said to be untoward.

The information of greatest value in forming an esti-

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<sup>6</sup> A letter by C. W. CRAMPTON in the *Jour. Amer. Med. Assn.*, 76, 16: 1121 (April 16, 1921), contains references to pertinent articles on this subject.

mate of a patient's heart is whether it permits him to carry on his daily work without symptoms. Any diminution in the capacity to perform work to which the patient has been habituated requires explanation. Questioning as to the effect of ascending stairs or walking on the street often elicits helpful information.

The exercise tolerance should rarely be tested in the presence of fever and it is unnecessary in the presence of definite evidence of cardiac failure. It should be reserved for cases in which some doubt exists as to the presence of cardiac disease, or as an important aid to assessing the amount of impairment of the heart, and a useful guide to treatment.

Thomas Lewis,<sup>7</sup> in his usual lucid manner, summarizes the interpretation to be placed upon a deficiency in the exercise tolerance somewhat as follows:—

1. In the presence of definite enlargement of the heart, aortic disease, mitral stenosis, or fibrillation of the auricles, any undue distress after exercise should be attributed to cardiac disease.
2. In young subjects, if none of the above four conditions are present, a deficient exercise tolerance should rarely be attributed to the heart. Tuberculosis and other pyogenic infections are likely.
3. In the elderly, in whom there is no sign of structural change, if the exercise tolerance is poor, the heart should be blamed. Such persons are liable to suffer from angina pectoris or myocardial damage, in which the chief symptoms may be breathlessness or undue fatigue, rather than pain.

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<sup>7</sup> "Cardinal Principles in Cardiological Practice," *Brit. Med. Jour.*, 2: 621 (Nov. 15, 1919).

RÖNTGEN EXAMINATION.—This is primarily the province of the radiologist and, therefore, its discussion will be limited to some of the points thereon. The Röntgen findings are particularly useful in giving us the most accurate data as to the shape and size of the heart and aorta, and to the detection of aneurisms, etc.

The patient should first be examined under the fluoroscope and the general contour of the heart and great vessels observed. Any abnormal pulsations should be noted, and, with experience, considerable can be concluded as to the heart rhythm by watching the contractions of that viscus. Examination with the patient standing at oblique angles should not be omitted, as without this procedure, certain aneurismal changes of the aorta may escape detection.

One or more exposures are then taken, and the negatives studied for details. These are usually taken at a distance sufficient to permit mensuration of the heart and aorta. Seven feet is the standard distance in many clinics, but it can be shown that the margin of error at a four-foot distance is small. In other words, it is possible to study the size of the heart in plates taken at a distance of four or more feet; below this distance the possible distortion in the size of the image is too great.

Tables of the measurements of hearts in normal men have been published by several radiologists, but these have failed to be of the value that was hoped. The difficulty is that there is no normal-sized heart; the heart fits the individual in whom it is placed. The variations in the different measurements amount to several centimetres. Of late there has been an attempt to use the ratio of the transverse diameter of the heart to the internal transverse diameter of the chest. Danzer<sup>8</sup> found this ratio to vary in the

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<sup>8</sup> DANZER, C. S.: *Amer. Jour. Med. Sci.*, 157: 520, 1919.

normal from 39 to 50 per cent. If the ratio was 53 per cent. or higher, the heart was definitely pathological. Even when the ratio does not exceed 50 per cent., cardiac hypertrophy cannot be ruled out; for example, in the drop heart of ptosis, judgment must be guided by the intrinsic measurements.

The orthodiagnostic method of determining the size of the heart is complicated, but offers some advantage over the method, teleradiography, discussed above. Its description will be omitted.

The contour of the heart, as shown on the Röntgen film, often enables the radiologist to make a correct diagnosis of the existence of valve lesions. Thus, when he writes, "The heart is enlarged transversely, and is particularly prominent in the region of the auricles," he is likely to add, "The picture is that of mitral stenosis." Hypertrophy of the left ventricle is recognized by extension of the shadow downward and to the left.

The shadow of the great vessels may be enlarged or abnormal in contour. Broadening may be due to syphilis, arteriosclerosis, hypertension, rheumatic heart disease, high diaphragm, or even a dilated pulmonary artery. These conditions should be remembered before deciding, on Röntgen evidence alone, that an increase in width of the shadow of the great vessels is sure evidence of luetic infection of the aorta. This matter and the occurrence of aneurisms will be dwelt upon further in subsequent chapters.

**VITAL CAPACITY.**—This is determined by measuring the volume of air which is expressed in a forced expiration. The patient exhales the maximal amount of which he is capable, into a spirometer, and the content of air is read from the scale on the instrument.

The actual value of the method is gradually being

established. Occasionally there is an evident lack of co-operation on the part of the patient. A decrease of the vital capacity may be due to other causes than heart disease, for instance, pulmonary affections. An authority on this method of examination well expresses its present status as follows: "In heart disease the vital capacity is of practical value after the diagnosis has been established and as a direct index of cardiac reserve... The actual change in any given case is of more importance than the change in percentage of vital capacity as compared with normal standards. In general, physical training may increase the vital capacity about 25 per cent. above the normal, and extreme physical weakness may reduce the vital capacity about 25 per cent. below the normal standards."<sup>9</sup>

The chief reliable signs of heart disease have been listed by Lewis,<sup>10</sup> as follows:

- (a) An aortic diastolic murmur.
- (b) Distinct over-distention of the veins of the neck.
- (c) Definite signs of enlargement of the heart.
- (d) An irregular heart action which is maintained on exercise (the heart rate being high).
- (e) A diastolic rumble at the apex.
- (f) A basal or apical thrill. The thrill must be an unmistakable "purr;" a suspicion of thrill is insufficient.
- (g) Widespread arterial disease or a persistent blood pressure, 180 or over in an elderly man, arterial disease or a persistent blood pressure of 160 or over in a young man.

<sup>9</sup> PEABODY, F. W.: *Med. Clin. No. Amer.*, 4, 6: 1655 (May, 1921).

<sup>10</sup> LEWIS, T.: *The Soldier's Heart and the Effort Syndrome*, 1919, Paul B. Hoeber, New York, p. 132.

## CHAPTER III

### METHODS OF EXAMINATION (cont.)

#### Graphic Methods

THE graphic methods are the examinations with the polygraph and the electrocardiograph. The use of these instruments of precision has already become a valuable addition to clinical cardiology. They will be described in order. The reader may prefer to defer reading these chapters until reaching the section on Functional Conditions. Without their assistance, however, it is doubtful if the various arrhythmias will be clearly understood.

#### POLYGRAPHIC EXAMINATION

The brilliant studies of the venous pulse by James Mackenzie, supplemented by those of Wenkebach and others, have made possible the understanding of many cardiac conditions hitherto obscure. The taking of pulse tracings is now widely practised. Besides its value in diagnosis, a polygram serves as a permanent record of certain conditions which at best can be but imperfectly described by words. The information obtained is somewhat less than that produced by electrocardiography, save in the matter of pulsus alternans, which is best demonstrated by a tracing of the arterial pulse. The polygraph possesses the additional advantage that it may readily be carried to the patient's bedside and thus used when an electrocardiographic examination is unobtainable.

There are several sphygmographs and polygraphs to choose from, but perhaps the best known, in this country, are the Mackenzie ink polygraph and the Sanborn instru-

ment. Both of these polygraphs (many writings) permit the simultaneous recording of any two pulsations. The tracings are registered by pens writing in ink on a strip of paper fed at chosen speeds by a clockwork mechanism. A third pen records the time in fifths of a second. Detailed description of these instruments will be omitted; prospective owners will find sufficient information in the descriptive leaflets supplied by the manufacturers.

It must not be thought that clinical polygraphs reproduce accurately the course of events inside the vessel under examination. Their vibration frequency is not great enough,<sup>1</sup> and, on account of their weight, the writing pens tend to override the mark. This results in an exaggeration of the height of the waves and often causes purely artificial secondary oscillations on the downstroke. Only the major waves are recorded, and these are given unnaturally rounded outlines, while the finer waves are slurred over or combined.<sup>2</sup> Too great friction of the pens against the paper introduces further mechanical defects. In spite of these limitations the pulse tracings are of value, due to the system of interpretation by means of the time relations between the arterial and venous curves, so extensively developed by Mackenzie, Wenkebach and others.

The taking of pulse tracings is a most fascinating study, but will prove disappointing unless the instrument is kept in good condition, especially as regards the renewal of the rubber of the tambours. Care and the use of much patience are often needed. The tracings are of uncertain value unless the examiner acquires a knowledge of their

<sup>1</sup> WIGGERS, C. J.: *Circulation in Health and Disease*, Lea and Febiger, Philadelphia and New York, 1915, p. 109.

<sup>2</sup> LEWIS, T.: *The Mechanism and Graphic Registration of the Heart Beat*, Paul B. Hoeber, New York, 1920, p. 21.

interpretation. Such knowledge may be obtained from the literature and by personal instruction. If approached in this manner the polygraph will become an instrument affording considerable pleasure and information pertaining to the heart, and will not be discarded after a few weeks.

The arterial curves are obtained by applying the receiving apparatus over the radial or brachial artery and adjusting until the writing pen registers the maximum excursion. If the pulse is of very small volume it may be recorded with difficulty.

In taking a tracing from the jugular vein it is usually essential that the patient be lying flat or nearly so, as otherwise the "a" or auricular wave may not appear at the neck. The cup is usually applied over the jugular bulb which lies beneath the angle between the clavicle and the posterior border of the sterno-mastoid muscle and the pressure adjusted until the optimal record of the waves is obtained. At times the cup must be moved to adjacent positions; in some cases a more satisfactory tracing may be obtained from the external jugular vein where it courses more posteriorly in the neck. The presence of severe dyspnoea with marked inspiratory retraction of the cervical tissues is an obstacle to the recording of a successful venous curve.

In nervous children or in patients who, because of delirium or some other reason, will not keep quiet, it may be a waste of time to attempt to use the polygraph.

The limitations set for this book make it possible to present but an outline of the study of pulse tracings. Those interested in further details of polygraphy should consult the literature.<sup>3</sup>

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<sup>3</sup> One of the more recent books is, *Graphic Methods in Heart Disease*, by HAY, JOHN; Oxford Medical Publications, London, 1921.

## MODERN CONCEPTION OF HEART DISEASE ARTERIAL PULSE TRACING

The arterial pulse curve, or sphygmogram, is used for the detection of pulsus alternans, the study of respiratory variations in volume, and in combination with the venous curve forms the polygram. Study of the arterial tracing often permits identification of the type of disordered heart action; the proof, however, usually requires the polygram or electrocardiogram.

**PULSUS ALTERNANS** is shown by a sphygmogram in which large and small waves alternate; the waves must be evenly spaced or the small waves may show slight delay, that is, in the tracing the small wave is further from the larger wave that precedes than from the one that follows it. The electrocardiogram and other evidence have shown that the contractions of the heart occur at equal intervals of time; the delay of the small wave is due to the fact that with the smaller output of blood there is a delay in the rise of aortic pressure and in the registration of the pulse wave. True pulsus alternans is to be distinguished from certain types of bigeminal pulse, due to the occurrence of an extrasystole after every normal beat, by the fact that in this pseudo-alternation, the longer pause follows the smaller wave, *i.e.*, the latter is premature.

Pulsus alternans is usually more prominent and sometimes detected only in the beats immediately following an extrasystole or a slight increase in the ventricular rate. Alternation is common in the accelerated pulse beats of paroxysmal tachycardia or flutter.

**RESPIRATORY VARIATION** in the strength of the arterial pulse waves is indicated where this occurs synchronous with the phases of respiration. For absolute proof, a respi-

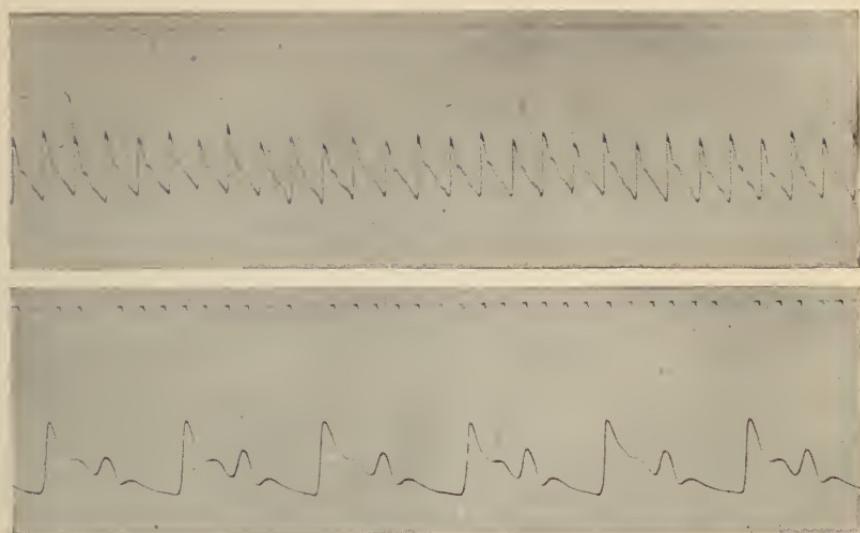


FIG. 5.—Upper tracing. Pulsus alternans. Lower tracing. Ventricular premature beats causing pseudo-alternation of the pulse. The small waves, which indicate the extrasystoles, are nearer to the preceding large waves. The paper is travelling at a more rapid rate than in the tracing of pulsus alternans. (The interpretation of these and the succeeding polygrams has been checked by electrocardiograms.)

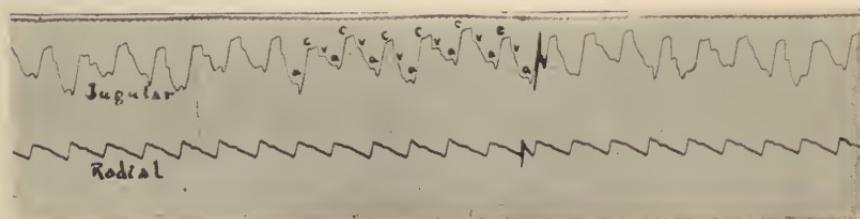


FIG. 6.—Normal polygram.

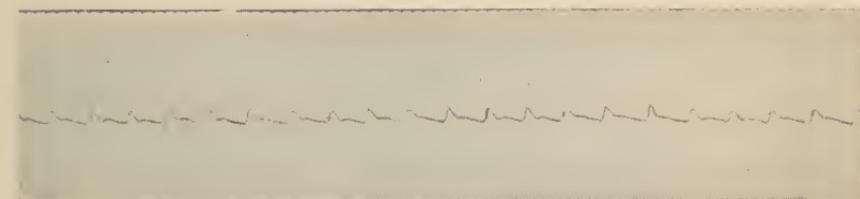


FIG. 7.—Sinus arrhythmia. Note alternate acceleration and slowing of the pulse.



tory tracing, or pneumogram, should be taken simultaneously. A respiratory variation in the pulse volume is often associated with the alternate acceleration and slowing of the pulse rate that occurs in sinus arrhythmia. Inspiratory weakening in the volume of the pulse is often termed pulsus paradoxus, or Kussmaul's pulse; while it may be present in adherent pericarditis, it has been repeatedly demonstrated that it may be without pathological significance.

**DOMINANT RHYTHM.**—The first step in the analysis of tracings of an irregular arterial pulse is to determine the presence or absence of a dominant rhythm. The latter may be defined as a rhythm which governs more or less the contractions of the ventricle. Usually the dominant rhythm arises in the sino-auricular node, but occasionally in some other part of the heart.

In an arteriogram of an irregular pulse, those waves which are preceded by the longest pauses and are greatest in amplitude are usually initiated by the dominant rhythm. A run of four or more regular beats, if these are the most forcible seen, reveals the presence of a dominant rhythm. The latter is displayed if the phases of irregularity are repeated, however irregular such phases may be; the presence of a controlling rhythm may be assumed whenever an irregular pulse shows periodicity of any kind.<sup>4</sup> When the dominant rhythm is undisturbed, a pulse irregularity is due to heart-block or extrasystoles arising in the ventricles.<sup>5</sup>

The determination of a dominant rhythm in an irregular arterial pulse is of importance in excluding an arrhyth-

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<sup>4</sup> LEWIS, T.: The Mechanism and Graphic Registration of the Heart Beat, p. 134.

<sup>5</sup> Exceptionally in the auricle.

mia due to fibrillation of the auricles. Since the identification of the different types of arrhythmia is greatly facilitated by the addition of data from the simultaneously recorded phlebogram, and this is readily available by the use of the polygraph, discussion of the subject may perhaps be more profitably continued if the evidence from the venous tracing is combined with that from the arterial.

### VENOUS CURVES

Tracings from the jugular vein present a succession of peaks and summits often arranged in a bewildering fashion. The curves are so variable that their interpretation should not be attempted without reference to an accompanying arteriogram. A number of waves may be present.

ABSCISSÆ or index marks should be present in both the venous and arterial curves. They are lines in the tracing made by stopping the clockwork of the polygraph and allowing the pens to inscribe an upright line on the paper while stationary.

The "C" Wave<sup>6</sup> represents the onset of ventricular systole. It may be identified by measuring the distance from any radial upstroke to an abscissa and transferring the measurement to the venous curve after correcting for the time required for transmission from the level of the neck to the wrist. If the arterial curve is obtained from the radial artery it has been shown that a correction of 0.1 second is needed, while if it is from the brachial artery, as is usual with the Sanborn polygraph, the correction is

<sup>6</sup>An excellent description of the fundamentals of the venous pulse will be found in "The Venous Pulse," EWING, E. M.: *Amer. Jour. Physiol.*, xxxiii:158, 1914.

nearer one half as much, or 0.05 second. The distance to be allowed for this correction is determined by comparison with the intervals inscribed by the time marker. The latter records periods of 0.2 second and therefore the correction is one half or one quarter of these spaces, according as the arterial tracing is from the radial or brachial artery, respectively. As the "c" in the neck occurs earlier than the arterial upstroke, the correction should be added to the arterial measurement if the latter is taken to the left of an abscissa (*i.e.*, the abscissa being on the right) and subtracted if the measurement is obtained from the right side of an abscissa.

A convenient method of identifying a series of "c" waves is to mark a series of radial or brachial upstrokes and the adjacent abscissa on the edge of a strip of paper. The latter is then transferred to the venous curve and moved to the left or right of the corresponding abscissa according to the correction (described above) that is needed. The marks will then underlie the beginning of the "c" waves. The "c" or carotid wave is so called as it was thought to be due to the impact of the carotid artery, and while in the neck it is synchronous or nearly so with the carotid pulsation, it has been shown to be present in the vein proximal to the beginning of the carotid artery.

The "V" WAVE.—The end of ventricular systole is represented in the jugular curve by the apex of the "v" or ventricular wave, which is probably due to stasis. The "v" wave is synchronous with the bottom of the dicrotic notch in the radial tracing. It may be identified by transferring to the venous tracing the measurement between the abscissa and bottom of the dicrotic notch in the arteriogram. In insensitive curves the dicrotic notch may not be

well represented, and in this case experience has shown that a sufficiently close estimate of its position may be made about two-fifths of a second after the "c" wave; absolute accuracy is not needed for this point. If desired a strip of curves from the carotid artery may be taken and the length of a beat transferred to the jugular tracing. Care should be used not to mistake a notch immediately following, in some tracings, the arterial upstroke for the dicrotic notch; it is earlier than the latter and is an artifact due to overshooting of the pen.

It is important to determine the "v" waves in polygrams; they may be mistaken for the "a" waves, or may coincide with the latter, in which case an exaggerated wave usually results. The "v" wave may be notched or insignificant.

The "A" WAVE.—The auricular wave is found directly before the upstroke of "c," and represents the auricular contraction in the venous curve. Normally it should be about 0.2 second before the onset of the carotid wave. A series of equidistant waves located in the diastolic phases of the phlebogram are usually "a" waves. If the wave preceding the "c" wave is prominent, it is the more certain to be the true auricular wave. When identification of "a" is dubious further evidence should be obtained from a tracing of the cardiac apex or from the electrocardiogram. According to Lewis,<sup>7</sup> "It is a golden rule to accept only those curves in which the interpretation is transparent, or in which repetitions in detail are secured."

The "H" OR "B" WAVE.—This wave is found in early diastole in some venous tracings of a slow rhythm. It

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<sup>7</sup> LEWIS, T.: The Mechanism and Graphic Registration of the Heart Beat, p. 142.

occurs synchronous with or a little after the third heart sound. Clinically it is unimportant.

**The A-C INTERVAL.**—This is the period between the upstrokes of the corresponding waves. It is used as an index of the auriculo-ventricular conduction period, though it is usually longer (not more than 0.1 second) than the P-R interval of the electrocardiogram. Normally the a-c interval varies from 0.1 to 0.2 second; a lengthening to 0.3 second or more indicates defective conduction between the auricle and ventricle.

The VENTRICULAR FORM OF VENOUS PULSE is represented by the absence of distinct "a" waves and the occurrence of the chief summits within the limits of ventricular systole: the AURICULAR FORM OF VENOUS PULSE is characterized by the presence of the "a" waves in addition to those associated with the contraction of the ventricle. Care must be taken that the absence of the "a" wave is not due to an error in the technic of taking the tracing from the neck. Thus the auricular wave will often not be recorded unless the patient is lying approximately horizontal, or if the receiving cup is placed over the carotid artery rather than in the positions described above. If the heart rhythm is regular, the ventricular form of venous pulse may be due to engorgement of the auricle, simultaneous contraction of auricle and ventricle, or regular action of the ventricle with auricular fibrillation. The ventricular form of venous pulse, associated with an irregular rhythm of the heart, is evidence of auricular fibrillation.

**SINUS ARRHYTHMIA.**—The polygram shows that all waves are present and in normal relation to each other, but they display a rhythmic acceleration and slowing. The change in rate usually corresponds to the phases of respira-

tion. Variation in volume is also often present, as already mentioned in the discussion of the arteriogram. A less frequent form is the so-called phasic type of sinus arrhythmia, in which the acceleration or slowing persists for a longer period than that of inspiration or expiration.

**SINO-AURICULAR BLOCK.**—This relatively infrequent arrhythmia is less readily recognized in the polygram. An apparently normal tracing in which the frequency of the waves is suddenly reduced to half, or approximately half, is a common form. At times a single beat may be dropped, as in some cases of auriculo-ventricular heart-block, but distinguished from the latter by the fact that the "a" wave disappears as well as the "c" and "v" excursions.

**PREMATURE BEATS OR EXTRASYSTOLES** may arise in the auricle or in the ventricle, and exceptionally in the A-V node. In the auricular type the ventricular waves ("c" and "v") are preceded by an "a" wave in the venous curve, while in the ventricular type the "a" is absent. The premature beat may or may not appear in the arterial tracing according as the heart expels an amount of blood sufficient to make a wave in the artery. If the "a" wave of the premature auricular beat happens to coincide with the "v" or "c" excursions of the ventricular contraction in response to the previous auricular systole, a wave of exaggerated height occurs due to the superimposition of the two waves. A further explanation of these amplified waves is that the force of auricular systole is felt more strongly in the jugular vein when the auricle contracts against a closed auriculo-ventricular valve.

Premature beats are almost always<sup>8</sup> followed by a pause, and by noting the length of the latter it is often

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<sup>8</sup> Interpolated extrasystoles are a rare exception.

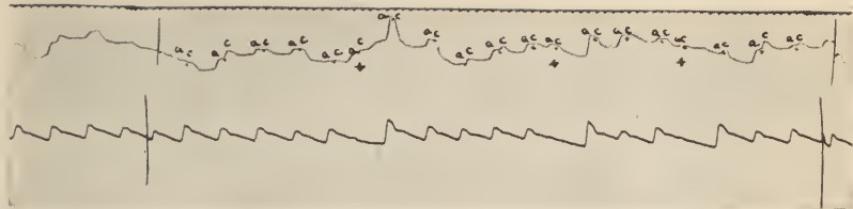


FIG. 8.—Auricular premature beats.

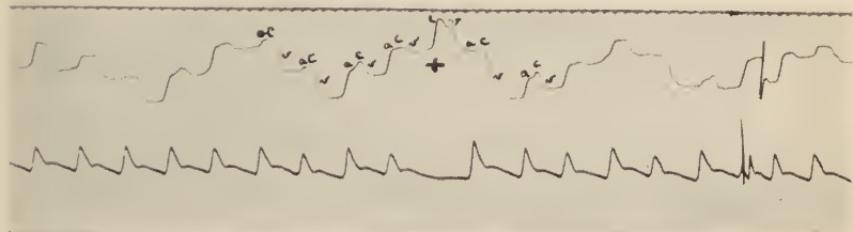


FIG. 9.—Ventricular premature beat.

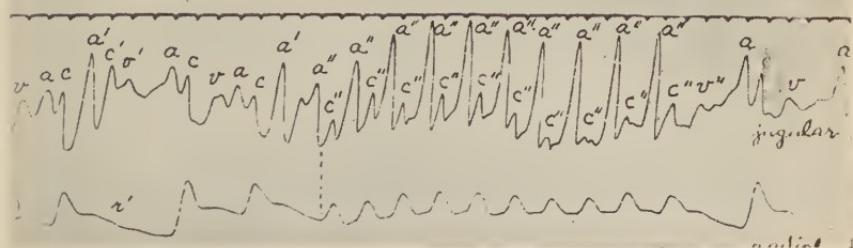


FIG. 10.—Paroxysmal tachycardia of auricular origin. The paroxysm has a duration of but ten beats, an auricular premature beat is recorded in the first part of the tracing, and the rapid rhythm is followed by the characteristic post-paroxysmal pause. (After Sir Thomas Lewis.)

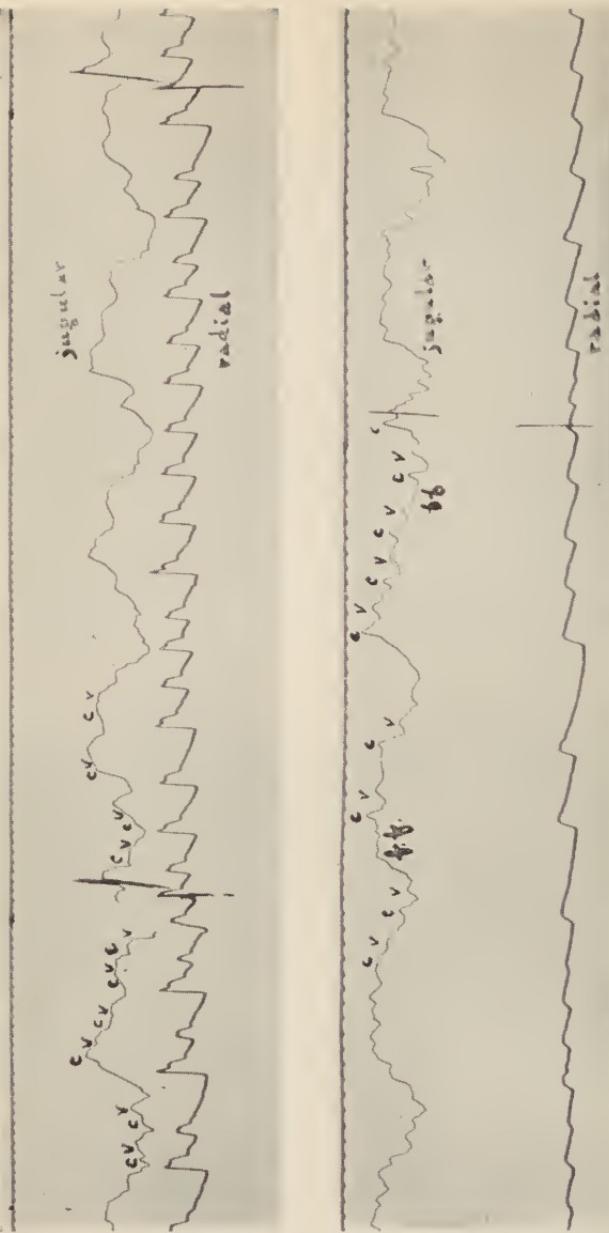


FIG. 11. Auricular fibrillation. Lower tracing taken at a greater speed of the paper. Some  $f$  waves are marked.

possible to determine the origin of the beat. The pause following the ventricular extrasystole is always fully compensatory, *i.e.*, of such a length that the succeeding beat of the normal rhythm occurs at exactly the time it would be expected if no extrasystole had occurred, and the dominant rhythm is not disturbed. When the pause is compensatory it will be found that the cycle of the premature beat and its succeeding pause is just equal to the sum of two normal heart cycles. The pause following a premature beat of auricular origin varies; it is usually longer than that after the normal beats and less than fully compensatory, but it may be equal to the normal pause, and rarely it may even have a duration that is fully compensatory. The variation in the post-extrasystolic pause is dependent on the distance of the new impulse centre from the sino-auricular node, from which the dominant rhythm starts, and the amount of the prematurity of the extrasystole. In the case of auricular extrasystoles followed, as is nearly always true, by a less than compensatory pause, the succeeding beat comes before it would have come if the premature beat had not occurred and the dominant rhythm is disturbed. In brief then, if the extrasystole disturbs the dominant rhythm it may confidently be ascribed to the auricle, whereas, if there is a compensatory pause so that the dominant rhythm is not disturbed the origin of the extrasystole is usually ventricular, but it may exceptionally be auricular.

The nodal extrasystole is not common. It gives rise to an exaggerated wave in the phlebogram due to the simultaneous contraction of the auricle and ventricle; the succeeding pause is usually compensatory. Extrasystoles originating in the A-V node are much more clearly identified in the electrocardiogram.

In cases of complete auriculo-ventricular heart-block, in which the ventricle is beating in response to an impulse centre in its own tissues, ventricular extrasystoles may occur and they interrupt this dominant rhythm exactly as auricular premature beats do the rhythm from the sino-auricular node. This combination is, however, of infrequent occurrence.

Interpolated extrasystoles are ventricular contractions which occur prematurely and in which the retrograde progress of the impulse to the auricle is blocked in the auriculo-ventricular bundle. In this case the rhythm of the auricle is undisturbed; the ventricle also contracts in response to each auricular impulse. The interpolated ventricular contraction may be said to be the only extrasystole which is literally an extrasystole. It is not common.

**PAROXYSMAL TACHYCARDIA.**—The arteriogram shows a series of rapid and regular beats which may be so close together that the succeeding beat starts on part of the dicrotic wave and gives an anacrotic effect. In the jugular curve the “a” wave may coincide with part of the preceding ventricular systole and cause the wave to be superimposed. In this manner the ventricular form of venous curve may result. It is frequently not possible to determine from the polygram whether the tachycardia originates in the auricle or in the ventricle unless the tracing shows the onset or cessation of the paroxysm. In that case it is usually possible to identify and establish the origin of a premature beat which initiates the attack and may often be seen at its termination. Alternation of the arterial waves is common in paroxysmal tachycardia. At times it is difficult to obtain satisfactory polygraph tracings due to the small volume of the pulse.

**AURICULAR FLUTTER.**—In this condition the auricular rate averages about 300 per minute and the waves in the venous pulse are often too feeble to be recorded, in other cases it is difficult to disentangle them from the ventricular waves, and at times the phlebogram may closely simulate a normal jugular tracing.<sup>9</sup> The arteriogram may be regular or irregular; in the latter case it can be established that a dominant rhythm is present. The signs of a dominant rhythm have been discussed above, but the method of spacing, by which the irregular arterial tracing of flutter is interpreted in relation to the auricular rhythm, is complicated and cannot be clearly presented in the limits set for this chapter. The interested reader is referred to the larger books of Lewis<sup>9</sup> and others. The diagnosis of auricular flutter usually requires confirmation by the electrocardiogram, and in this, fortunately, the evidence is nearly always clear.

**AURICULAR FIBRILLATION.**—In the arterial tracing the picture of absolute arrhythmia is presented; a dominant rhythm is absent. The venous curve discloses the absence of the "a" waves, *i.e.*, it is the ventricular form of venous pulse. In the cases of long standing in which considerable dilatation of the auricle is present the venous curve shows an ascent during the longer diastoles and on this may be seen the "ff" waves. This interesting detail consists in small oscillations, usually regular. The "ff" waves apparently are found in no other condition.

**HEART-BLOCK.**—In incomplete heart-block the a-c interval is prolonged beyond 0.3 second. In the venous tracing this may cause the "a" wave to coincide with the

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<sup>9</sup> LEWIS, T.: The Mechanism and Graphic Registration of the Heart Beat, p. 267.

ventricular waves (that follow the previous auricular impulse) and, by their superimposition, to produce an exaggerated excursion. Variations in the a-c interval are common. Sometimes the impulse may be blocked and then there is a gap in the arterial curve and the "a" wave in the phlebogram is not followed by the ventricular waves. There may be but half or a third or fourth as many up-strokes in the arterial curve as there are "a" waves in the venous tracing in which the ventricle is not beating in response to every auricular contraction, *i.e.*, when a 2-1, 3-1, 4-1, or higher rhythm exists. A change from a 1-1 to a 2-1 or higher rhythm will cause an irregularity in the arteriogram. If the arterial tracing is irregular in incomplete heart-block, examination of a sufficient length of the curve will show the presence of dominant rhythm. The irregular arteriogram of incomplete heart-block can be distinguished from that of flutter only by the assistance of a successful phlebogram or, better, by an electrocardiogram.

In complete heart-block complete dissociation exists. The ventricle contracts in response to an impulse centre in its own tissues, and this is represented in the arterial curve by a series of regular waves of a frequency of about 30 to 40 per minute, though sometimes higher or lower. In the jugular tracing the ventricular waves are present at their expected places in relation to the arterial curve, and between them occur other waves. These latter, if spaced regularly in relation to each other, may be identified as "a" waves. If, on determination of the rate of the "a" waves and that of the arterial beats, the latter are not in relation to the former in a ratio of 1 to 1, 2 to 1, or higher, the diagnosis of complete heart-block is confirmed. An

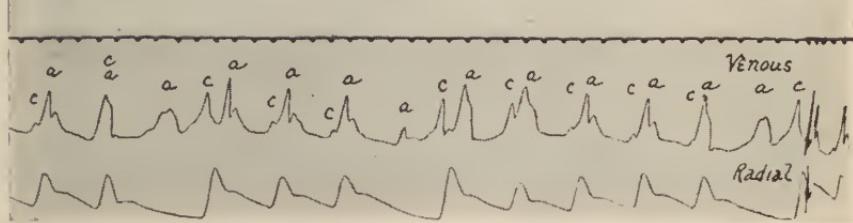


FIG. 12.—Partial heart-block. The *a-c* interval is prolonged and the third, seventh, and thirteenth *a* waves are blocked; the other *a* waves are exaggerated due to their superimposition on waves of the preceding ventricular systole. (After Sir Thomas Lewis.)

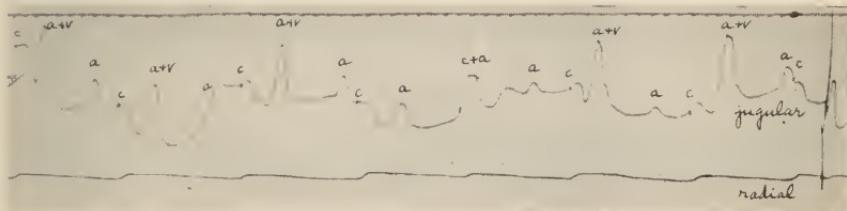


FIG. 13.—Complete heart-block. Auricular rate 61; ventricular 34; the two rhythms are regular but independent of each other. Where the *a* wave coincides with ventricular systole a conspicuous summit appears.



occasional dropped beat disturbs, of course, this numerical ratio, but the evidence that the "a" waves are in relation to the succeeding ventricular excursions in the venous tracing should make it apparent that the block, in such a case, is partial and not complete.

Polygraph tracings are also taken from the apex of the heart, the carotid artery, and over the liver, but their importance does not warrant description in this limited presentation of polygraphy.

## CHAPTER IV

### METHODS OF EXAMINATION (concl.) Graphic Methods (concl.)

## ELECTROCARDIOGRAPHIC EXAMINATION

A COMPLETE exposition of electrocardiography is beyond the scope of this book. The literature is extensive and the work highly technical; he who would fit himself to engage in electrocardiography should acquire a familiarity with its literature and spend considerable time in an electrocardiographic laboratory.

In the words of a master, Thomas Lewis; "These records have placed the entire question of irregular or disordered mechanism of the human heart upon a rational basis, so giving to the worker the confidence of knowledge; they have influenced prognosis and have rendered it more exact; they have potentially abolished the promiscuous administration of cardiac poisons, and have clearly shown the lines which therapy should follow. The new clinical observations have stimulated and directed a host of laboratory researches, anatomical, physiological, pathological, and pharmacological, of a valuable nature."<sup>1</sup>

Electrocardiography is of value in clinical medicine and the reader should be familiar with its essential facts. The electrocardiogram enables one to analyze 96 to 98 per cent. of the arrhythmias, which is perhaps 6 to 8 per cent. more than can be worked out by the tracings taken by the polygraph. It gives detailed information as to the origin and

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<sup>1</sup> The Mechanism and Graphic Registration of the Heart Beat, by T. Lewis. Paul B. Hoeber, New York, 1920.

passage of the contraction wave over the heart. It helps to elucidate many points observed clinically and on which the pathologist cannot offer any assistance. The various arrhythmias frequently do not display the typical picture and are then not easily diagnosed by the non-instrumental methods of examination, and when two or more of these functional conditions are present in combination, the problem is often too obscure for ordinary bedside diagnosis. The detection of impaired conduction in the ventricle and the accurate differentiation of the ventricular type of paroxysmal tachycardia from that of auricular origin, both of which are of distinct importance in prognosis, are dependent upon the use of the electrocardiograph. As regards which valve, if any, is affected, the electrocardiogram offers evidence of but indirect value and not infrequently is of no assistance. Electrocardiography may be said to have two distinct limitations: (1) it usually fails to demonstrate pulsus alternans, and (2) there are a few electrocardiograms which are difficult to interpret. With experience the latter tend to become rarer. The use of the electrocardiograph forms, of course, but one of the methods of cardiac examination, and to be most helpful, must be employed with the latter; it should not be allowed to lessen one's efforts to make accurate diagnoses without this aid, but its unmasking of our failures should react like the post-mortem examination, to guide and stimulate effort towards greater efficiency in diagnosis.

**HISTORY.**—It is known that when a muscle contracts, the contraction is accompanied by an electrical change. In 1855, Kölliker and Müller<sup>2</sup> first demonstrated, in the

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<sup>2</sup> KÖLLIKER, A., and MÜLLER, H.: *Verhandl. d. phys. med. Gesellsch. i. Wurzburg*, vi: 528, 1855.

heart of a frog, that an electrical current was associated with each of the contractions. The capillary electrometer was used to measure the current. In this instrument there is a column of mercury which moves up and down in a glass tube and may be photographed. The instrument is subject to the error of overshooting. In 1887, Waller<sup>3</sup> made the first use of the capillary electrometer on human beings. The first good records with the capillary electrometer were obtained by Bayliss and Starling,<sup>4</sup> in 1892.

The string galvanometer was introduced by Einthoven,<sup>5</sup> of Leyden, in 1903, to replace the capillary type. This new instrument is so superior that it has superseded other forms of sensitive galvanometer. The electrocardiograph remained, however, largely a laboratory instrument, of use to the physiologist but not to the clinician. About 1908 or 1909, Thomas Lewis, of Cardiff, Wales, became acquainted with the electrocardiograph in the laboratory of Edelmann, of Munich, and on his return to England, the Cambridge Scientific Instrument Company made him one to use. Lewis was fortunate in having the opportunity to examine patients in the unusual clinic which James Mackenzie had built up by his fame from his studies of the venous pulse, and in a short time the essentials of clinical electrocardiography were worked out. To-day the electrocardiograph is widely used in clinical medicine.

The American electrocardiograph, the Hindle, was started in 1917. This is now manufactured at Ossining, N.Y. The firm has associated with it Dr. Williams, a

<sup>3</sup> WALLER, A. D.: *Jour. Physiol.*, viii: 229, 1887.

<sup>4</sup> BAYLISS, W. M., and STARLING, E. H.: *Monthly Internat. Jour. Anat. and Physiol.*, ix: 256, 1892.

<sup>5</sup> EINTHOVEN, W.: *Annalen der Physik.*, Folge iv, xii: 1059, 1903.

physicist, who has contributed very largely to the high quality of the Hindle machine.

The best authorities on the physical side of electrocardiography are Einthoven, Lewis, and Williams.

**THE INSTRUMENT.**—This can be understood satisfactorily only by personal inspection. Its essential feature is a very fine fibre or string of quartz or glass made opaque by a coating of silver or platinum. This fibre is 0.002 to 0.005 mm. in thickness. It is suspended between two poles of a magnet and oscillates in accordance with the passage of the intermittent electrical current produced by the beating of the heart of the patient with whom it is in circuit. The string is illuminated by a powerful electric light, magnified (900 times in the Hindle instrument) by lenses of a microscope, and its oscillating shadow focussed on the front of a motion camera where it is photographed.

**ELECTRODES.**—As mentioned above, the electrical changes in the heart were first detected by placing the contacts or electrodes on the exposed organ; later it was found that records could be obtained from the external surface of the chest wall; and now it has been established that comparable results may be secured from electrodes placed on the limbs. The latter is the procedure in ordinary clinical work. The patient is connected with the string by means of three electrodes,—on the right forearm, left forearm, and left lower leg, respectively. A common type of electrode is a sheet of zinc covered with a pad wet with warm, twenty per cent., salt solution. Another form of electrode consists of a porous pot filled with warm salt solution and well-washed cotton wool; this is surrounded by a larger vessel containing a saturated solution of zinc sulphate in which a sheet of zinc is immersed, and to the

latter is soldered the leading-off wire. The patient's hands and left foot are placed in the salt solution of the inner vessel of the respective electrode.

**STANDARDIZATION OF THE ELECTROCARDIOGRAM.**—The purpose of this is that the records taken in different laboratories and on the same patient at different times may be kept to the same scale of excursion so that comparisons may be permissible. Einthoven's standard, in which one centimetre of excursion in the final photograph is equivalent to one millivolt potential difference at the end of the recording fibre, is now universally adopted. (The necessary qualifications of the electrodes, the technic of taking the electrocardiogram, and other details are fully discussed by Lewis in his book,<sup>6</sup> to which those desiring fuller information are respectfully referred.)

**THE LEADS** or derivations. There are three of these, made by a turn of a key on the switchboard of the electrocardiograph to connect any two of the electrodes on the patient. Lead I is between the two arms, lead II from the right arm to the left leg, and lead III between the two left limbs. The picture obtained varies in the three leads; identical waves are of different amplitude. This may perhaps be better appreciated by examination of Figure 14, the so-called Einthoven triangle. The three leads are indicated by the Roman numerals and respective limbs by the abbreviations R.A., L.A., and L.L. If the line A.B. represents the action current in the heart at a given moment, it will be represented in the respective leads as excursions of the size indicated by the lines A'B' placed on the sides of the triangle. In accurately taken electro-

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<sup>6</sup>The Mechanism and Graphic Registration of the Heart Beat, Paul B. Hoeber, New York, 1920.

cardiograms, it has been shown that the height of any individual wave in a single lead can be calculated from its values in the other two leads; for example, the height of a wave in lead II is equal to the sum of its excursions in lead I and III, etc.

Leads I and III are of value chiefly for the QRS complex and the determination of ventricular preponderance.

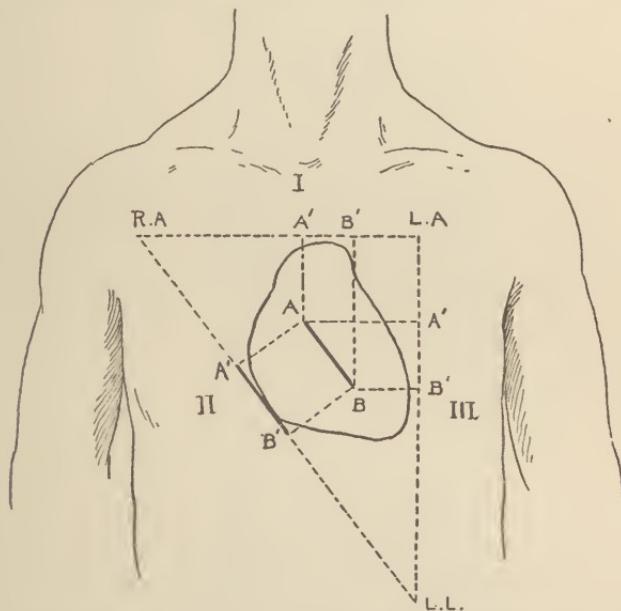


FIG. 14.—The Einthoven triangle, indicating diagrammatically the relation of the three leads to the heart.

Practically all other conclusions may be drawn from lead II, which is therefore the most important derivation. It will be noted that lead II is much more nearly parallel to the anatomical axis of the heart; the electrical and anatomical axes do not, however, coincide exactly.

**THE NORMAL ELECTROCARDIOGRAM.**—The electrocardiogram is a photograph of the oscillations of the string

of the galvanometer caused by changes in the electrical potential as the excitation wave passes over the heart. At first sight (Fig. 15), it looks like a zigzag line, but on closer study, it can be seen that the departures from the base line are repeated in a regular order. These departures from the base line have been named in an arbitrary manner (before their significance was known) the P, Q, R, S, T and U waves.

The waves are termed upright or inverted according as they are above or below the base line. When there is no current passing through the string, the image of the latter is flat, and the position is designated as isoelectric.

The background of the electrocardiogram is ruled into squares. The horizontal lines are known as the ORDINATES and are produced by lines engraved on the lens in the camera. They make it possible to measure the excursion of the waves from the base line and thus, in electrocardiograms taken in the standardized manner, to determine the electromotive force. The distance of each ordinate indicates an electromotive force of one tenth of a millivolt ( $0.0001$  volt) which is often written  $1^{10-4}$  volt. Every fifth ordinate is heavier as an aid to the eye. The vertical rulings are the ABSCISSÆ, which permit measurement of the waves in respect to their duration in time. The fine-lined abscissæ mark off periods of one twenty-fifth of a second, while the heavier ones, *i.e.*, every fifth, indicate one fifth (0.2) second. These abscissæ are made by the time marker, a revolving wheel, whose spokes, at the above intervals, cut across the stream of light in the electrocardiograph. The rate of the heart beat per minute is determined by counting the number of times some definite wave, usually the R wave, is repeated. A convenient method is to count

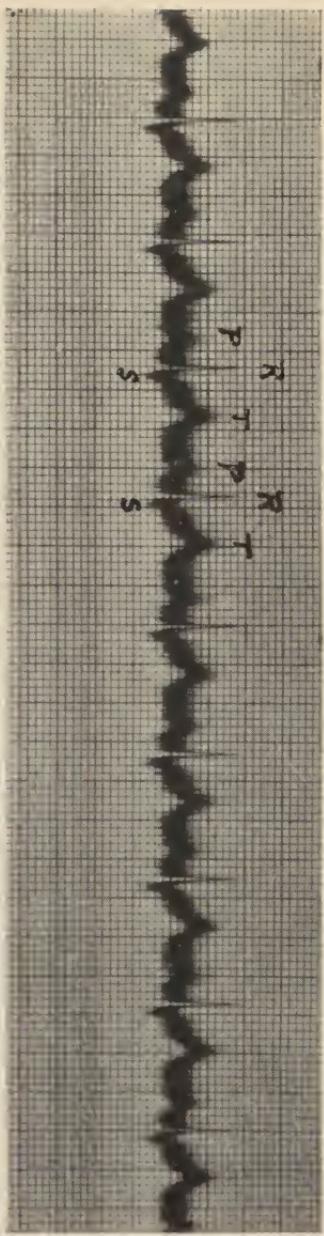


FIG. 15.—Normal electrocardiogram.\*

\*It is emphasized that this and the following small group of electrocardiograms are merely single examples of the various rhythms; all records of these rhythms are not necessarily identical in appearance.

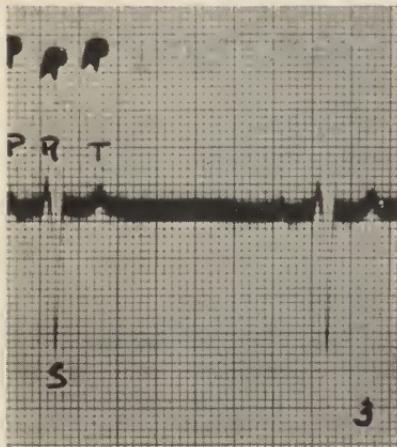
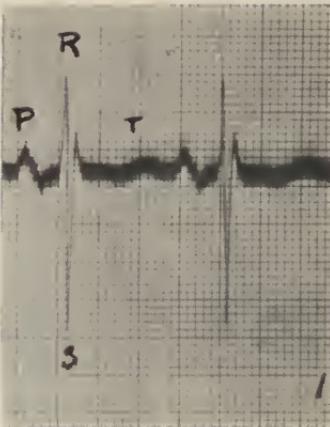
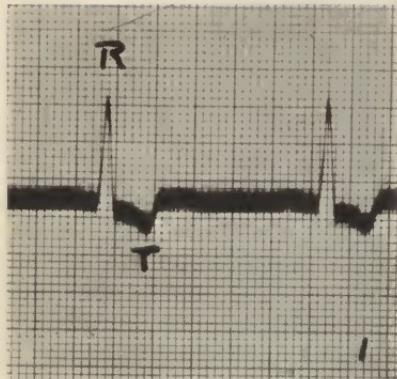


FIG. 16.—Ventricular preponderance.

Left electrocardiograms—leads I and III from a case of cardiovascular syphilis with aortic regurgitation. The index is +32.5, indicating left preponderance. The inverted T wave in lead I is a digitalis effect.

Right electrocardiograms—leads I and III from a case of rheumatic heart disease with mitral stenosis. The index is -27, indicating right preponderance.

the number of R waves occurring in six seconds (thirty successive fifths of a second in the tracing) and then to multiply this number by ten, for the rate per minute.

The P WAVE is associated with the contraction of the auricles. It must be upright in lead II, and not more than three tenths of a millivolt in force or more than one tenth of a second in duration. The occurrence of a notch in the P wave does not indicate abnormality. The P wave is usually upright in lead I but is often very small and may be flat or isoelectric; in lead III it may be diphasic (showing an excursion on both sides of the base line) or split. Inversion of the P wave in lead II indicates that the impulse did not start from the sino-auricular node; the more definitely inverted is the P wave, the nearer to the node of Tawara is the new impulse centre assumed to be.

The P-R INTERVAL indicates the period of conduction between the auricle and the ventricle. It is found by measuring from the initial deflection of the P wave to the initial deflection of the succeeding R wave. Normally, the interval varies from 0.12 to 0.18 second, though Lewis has recently extended its maximum length to 0.21 second. It is a safe rule that if the P-R interval exceeds 0.2 second, which is just equal to the space between the coarser lines made by the time marker, one is dealing with some degree of A-V heart-block.

The QRS COMPLEX is the result of the initial spread of the excitation wave in the ventricle. The Q and S WAVES may be small or absent and are relatively unimportant in ordinary clinical work.

The R WAVE is a sharp upward spike and is usually the most prominent wave in the electrocardiogram. It is abnormal if it has a duration of more than 0.1 second, or

if it is much thickened and notched in lead II. Simple notching is not infrequent in leads II and III in normal electrocardiograms.

The T WAVE is the end deflection of the ventricular complex. It is produced by the decline of the state of excitation in the ventricular muscle; but further than this its exact meaning is as yet somewhat obscure. Normally the T wave should be upright in lead II.

Flattening or inversion of the T deflection has been found associated with weakness of the myocardium and with hypothyroidism, and particularly as a sign of the action of the drug digitalis. The depression of the T wave due to digitalis (present in figure 23 lower curve, 24 upper curve, and 25) is of a progressive nature, the wave being first flattened and then inverted. The depression includes both the T wave and some of the string shadow just preceding. T of the normal ventricular complex is a combination of right and left ventricular effects; the former tends to make T negative; the latter, positive.<sup>7</sup>

The U WAVE is a small inconstant deflection which occurs just after the T wave, in slow-acting hearts. It is not well understood and appears to be unimportant.

The beginning of the first heart sound commonly coincides with the descending limb of the R wave, while the second sound commences at about the end of the T deflection. It has been demonstrated in experimental work<sup>8</sup> on dogs that the upstrokes of the P and R waves precede the evidence of muscular contraction in the auricle and ventricle, respectively, by about 0.02 to 0.04 second.

<sup>7</sup> WILSON, F. N., and HERMANN, G. R.: *Heart*, viii, 3:274 (May, 1921).

<sup>8</sup> The Mechanism and Graphic Registration of the Heart Beat, by T. Lewis. Paul B. Hoeber, New York, 1920, p. 51.

VENTRICULAR PREPONDERANCE.—This term is used to indicate relative increase in the weight of one ventricle as compared with that of the other. It is not synonymous with hypertrophy, as in the latter condition the whole heart may be hypertrophied, but if the balance is not disturbed there is no preponderance of right or left ventricle. In the careful study by Lewis and Cotton of cardiac hypertrophy (which will be considered in Chapter XVIII), it has been demonstrated that the presence in the electrocardiogram of the feature termed ventricular preponderance is very reliable evidence of an increase in weight of the ventricular muscle. Certain American investigators,<sup>9</sup> however, have found that a relation between the form of the electrocardiogram and the relative weights of the ventricles does not exist unless the whole heart weighs over 250 gm. and then the relation is not a close one.

Ventricular preponderance is diagnosed (Fig. 16) from the study of the QRS complex in the first and third leads. If R is high in lead I, and has a downward deflection in lead III, preponderance of the left ventricle is suggested; the reverse of these conditions indicates right preponderance. Certain authors have doubted the accuracy of the above generally accepted interpretation.

When preponderance is present, it is possible to obtain a numerical estimate of its extent, or the INDEX, by means of formula. Thus,  $(R_1 + S_3) - (R_3 + S_1) = \text{index}.$ <sup>10</sup> In this formula  $R_1$  is the height above the base line of the R wave in lead I, as measured by the millimeter ordinates on the electrocardiogram;  $S_3$  indicates the depth of the S wave in

<sup>9</sup> WILSON, F. N., and HERMANN, G. R.: *Jour. Amer. Med. Assn.*, 78, 23: 1839 (June 10, 1922).

<sup>10</sup> WHITE, P. D., and BOCK, A. V.: *Amer. Jour. Med. Sci.*, v, 156: 17, 1918.

lead III, etc. This is merely subtracting the right ventricular values from those of the left. An index of +20 to +30 usually indicates left preponderance, while above +30 is always left preponderance. Values of -15 to -18 are usually evidence of right ventricular preponderance, and beyond -18 is always so. The variation in the position of the heart as affected by the height of the diaphragm, respiration, etc., makes a difference in the above index and yet not enough to account for the higher figures. This matter of ventricular preponderance does not take into consideration the depth of the heart mass, and on theoretical grounds there is much that is unsound, and yet the conception proves useful.

**SINUS ARRHYTHMIA.**—The auricular and ventricular waves are present and in normal relationship to each other, but the former, the P waves, are irregular in their rhythm. It is an arrhythmia in which the irregularity involves the whole heart and is due to a failure of the pacemaker, *i.e.*, the sino-auricular node, to start an excitation wave at regular intervals. The electrocardiogram shows the usual normal waves, but the groups representing the individual heart cycles do not maintain the customary regular spacing. The usual form of sinus arrhythmia is that in which the waves show a rhythmic acceleration and slowing, in accordance with inspiration and expiration. Sometimes a simultaneous waxing and waning of the height of the R waves is seen, due to the respiratory shifting of the position (and so of the relation of the cardiac electrical axis to the lead in use) of the heart. A less frequent type, termed phasic, is similar save that the acceleration and slowing do not conform to the respiratory curve; they commonly extend over somewhat longer periods.

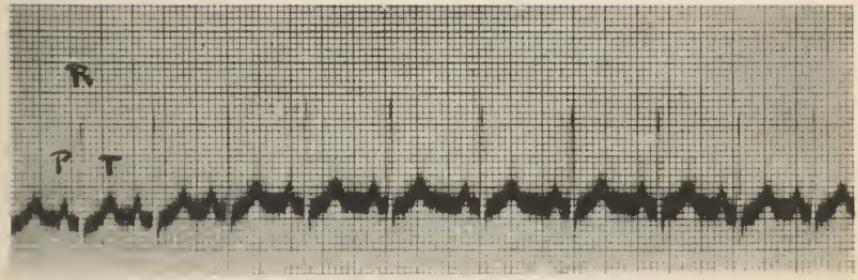


FIG. 17.—Sinus arrhythmia

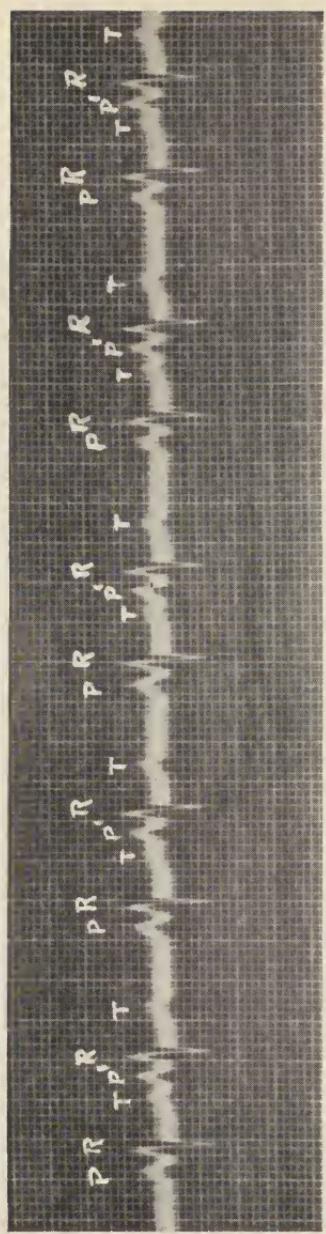


FIG. 18.—Auricular premature beats. Every other beat is premature; P' is slightly different in contour from the normal wave, marked P.

**SINO-AURICULAR BLOCK.**—In this condition some of the P waves appear to be absent or delayed. A common type of electrocardiogram is that in which the frequency of the waves is suddenly cut approximately in half. The P, R, and T waves are all present, but the above change in their frequency is the striking feature. In other instances a single or several beats appear wanting. This resembles the dropping of beats that takes place in certain cases of auriculo-ventricular heart-block, but is distinguishable from the latter by the fact that in sino-auricular block the P wave is missing as well as those of the succeeding ventricular complex. In many instances of sino-auricular block, the phenomenon of ventricular escape<sup>11</sup> occurs; in other words, if the stimulus to contract does not come from the sinus node in due time, the ventricle may beat in response to an impulse from a lower centre, usually the auriculo-ventricular node. The change of the place of origin from the sinus to the A-V node is often expressed as a shifting of the pacemaker. The electrocardiograms of instances of sino-auricular block may at times be quite complex and require care in their interpretation.

**ATRIO-VENTRICULAR RHYTHM or NODAL RHYTHM.**—When this is present the auricle and ventricle are contracting more or less simultaneously in response to impulses originating in the A-V node. The condition has been referred to in the discussion of sino-auricular block. The P wave is usually inverted, often distinctly so, but it is not always readily seen, as it may be buried in the ventricular complex. The contraction of the auricle may slightly precede, coincide with, or follow that of the ventricle; conse-

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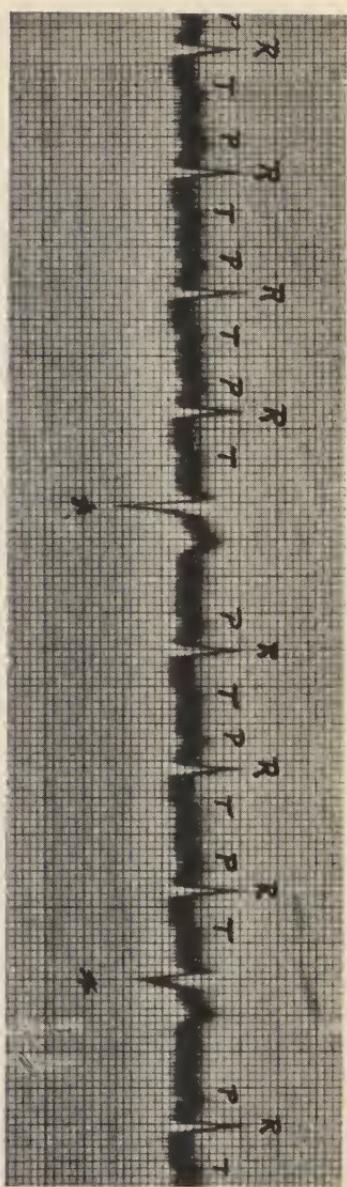
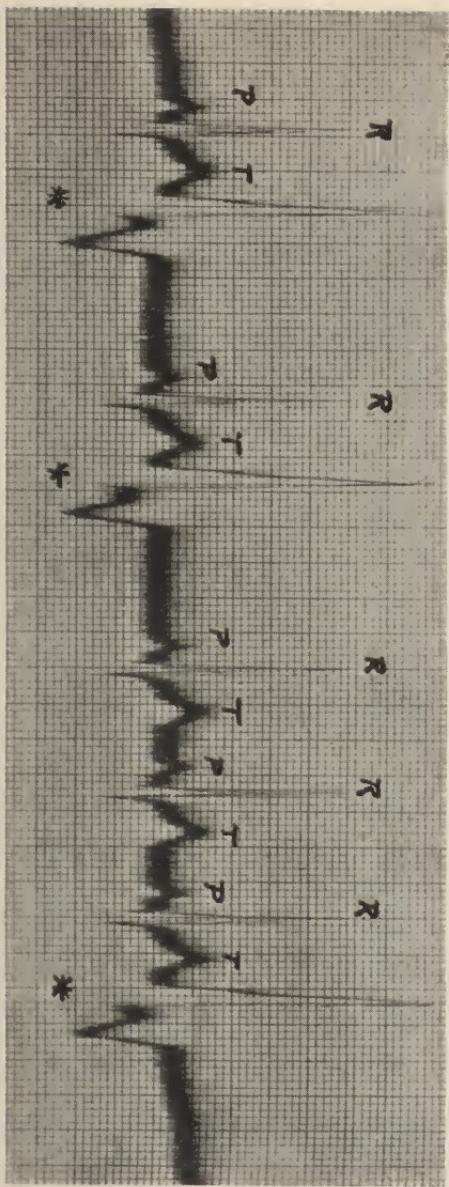
<sup>11</sup> Some authors have restricted the term to the response of the ventricle to an impulse originating on the ventricular side of the A-V node.

quently the P wave appears in these relationships to the ventricular complex of the electrocardiogram. Atrioventricular rhythm is not common; it is hardly to be diagnosed save by the graphic methods.

**PREMATURE BEATS, or EXTRASYSTOLES.**—These are heart beats which occur prematurely and do not form part of the rhythmic series of beats. They may arise from a focus in the auricle or in the ventricle, and occasionally in the A-V node. Their recognition is usually easy in the electrocardiogram. With the ventricular extrasystole, the ventricular complex is aberrant or abnormal in form, but in the case of the auricular and rarer nodal extrasystole, the succeeding ventricular complex is represented by normal waves. In exceptional instances, aberration of the ventricular waves may occur even though the extrasystole be of auricular origin; this is explained as probably due to impairment of conduction in the ventricular muscle, which has not fully recovered from the previous contraction. With the auricular extrasystole there is also evidence of the presence of a P wave, though the latter usually is different from the normal wave starting from the sino-auricular node. According to the degree of prematurity of the auricular extrasystole the P wave of the ectopic beat is advanced nearer to the preceding T wave, or it may be superimposed upon the latter. The variation in the length of the pause after the premature beats has been discussed in the preceding chapter; the length of the pause is of less importance in the electrocardiogram.

**PAROXYSMAL TACHYCARDIA.**—This arrhythmia may be defined as a regular tachycardia with an abrupt onset and cessation. Arbitrarily it is limited to regular tachycardias

FIG. 19.—Ventricular premature beats, interrupting normal rhythm. The ectopic beats are aberrant in form and are indicated by an \* placed below.



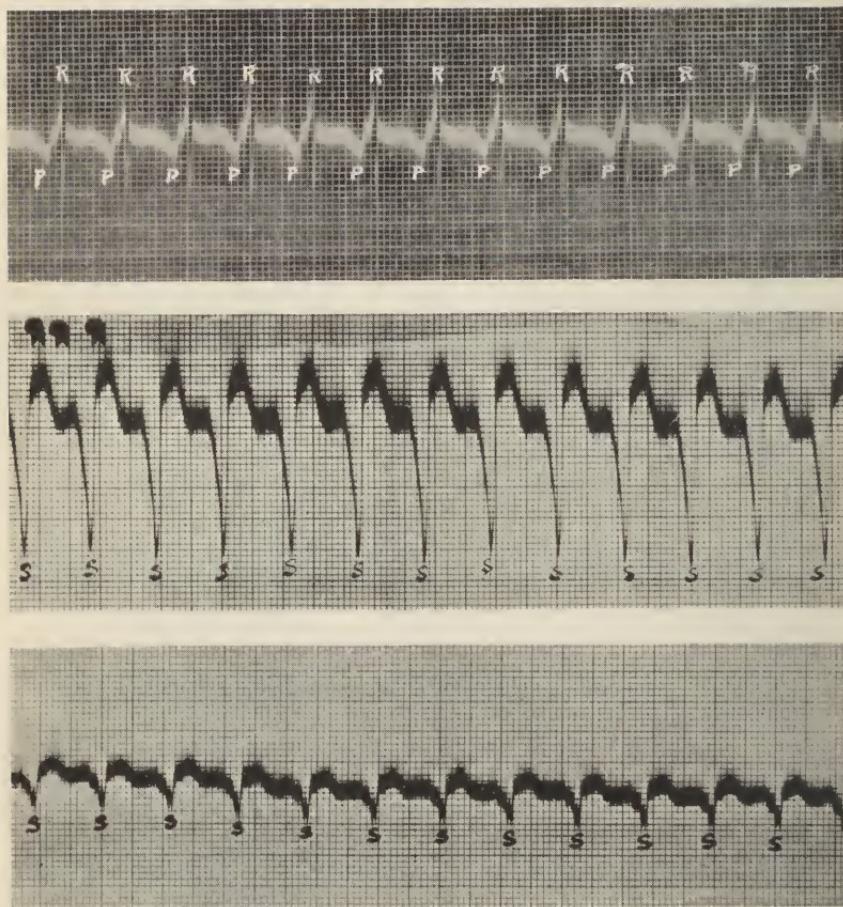


FIG. 20.—Paroxysmal tachycardia. Above, auricular type; centre, from ventricle; and below, of indeterminate origin.

not exceeding 200 per minute, though occasional cases of slightly more rapid rate are best assigned to this arrhythmia.

The paroxysm most commonly arises in the auricle, but may have its origin in the A-V node, or in the ventricle. Paroxysms of supraventricular origin nearly always have ventricular complexes of normal appearance; rarely, aberration may occur when the impulses rise in the auricle but follow an abnormal course in the ventricle. In the auricular type, the P wave is always abnormal, which is further evidence of its ectopic origin; usually it is inverted. In many cases the P wave is buried in the ventricular complex. If the electrocardiogram shows only the paroxysm, it is not always possible to determine the point of origin of the tachycardia; when the onset of the new rhythm is shown, the type of premature beat, of which the paroxysm apparently is a series, may be represented and its origin determined.

If the ventricular complex is aberrant (abnormal) in form, the paroxysm is probably of ventricular origin. Further evidence may be obtained from a study of the P waves, if present. An electrocardiogram showing the onset of the paroxysm may not be available, but if a record, taken when the attack is not present, discloses ventricular extrasystoles of which the ventricular complexes of the paroxysm are an exact replica (such was the case in the middle curve of Fig. 20), the ventricular origin of the new rhythm is almost certain. The ventricular type of paroxysmal tachycardia, unlike the commoner auricular form, is of distinctly bad prognostic import because of its frequent association with occlusion of the coronary artery and infarct of the myocardium. Robinson and Hermann<sup>12</sup> have em-

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<sup>12</sup> ROBINSON, G. C., and HERMANN, G. R.: *Heart*, viii, 59: 1921.

phasized the need of an electrocardiogram to make this important differentiation.

**AURICULAR FLUTTER.**—In this condition the auricle beats at a rapid and regular rate, which varies in human subjects from 200 to 365 per minute. The average rate is about 300. The electrocardiogram is important in the diagnosis of auricular flutter, as the latter can rarely be recognized save by the use of instrumental methods of examination, and the polygram is often unsatisfactory due to insufficient excursion of the waves. The auricular waves are represented by a continuous series of P excursions, giving to the picture of the string a regular zigzag appearance interrupted by the ventricular complexes. When the ventricle is beating at a rapid rate and in less favorable leads (lead I, and sometimes lead III) the superimposition of the ventricular waves on the series of P waves may obscure the regularity of the latter. The electrocardiogram shows constant activity of the auricle, but that of the ventricle is less frequent. Thus, 2 to 1, 3 to 1, or higher ratio maintains; in a certain sense auriculo-ventricular heart-block is present, but that the latter is not the primary or essential rhythm is readily evident from the abnormally high rate of the auricular waves. It is emphasized that the auricular waves show a high degree of regularity; those of the ventricle may be regular or irregular, the latter resulting from a variation in the rate of response to the supraventricular stimuli. The ventricle rarely responds to all of the auricular contractions and, if so, only for a short time; so high a ventricular rate is incompatible with the power of the ventricular muscle and the maintenance of the circulation.

**AURICULAR FIBRILLATION.**—In this frequent and important arrhythmia there is always some part of the auric-

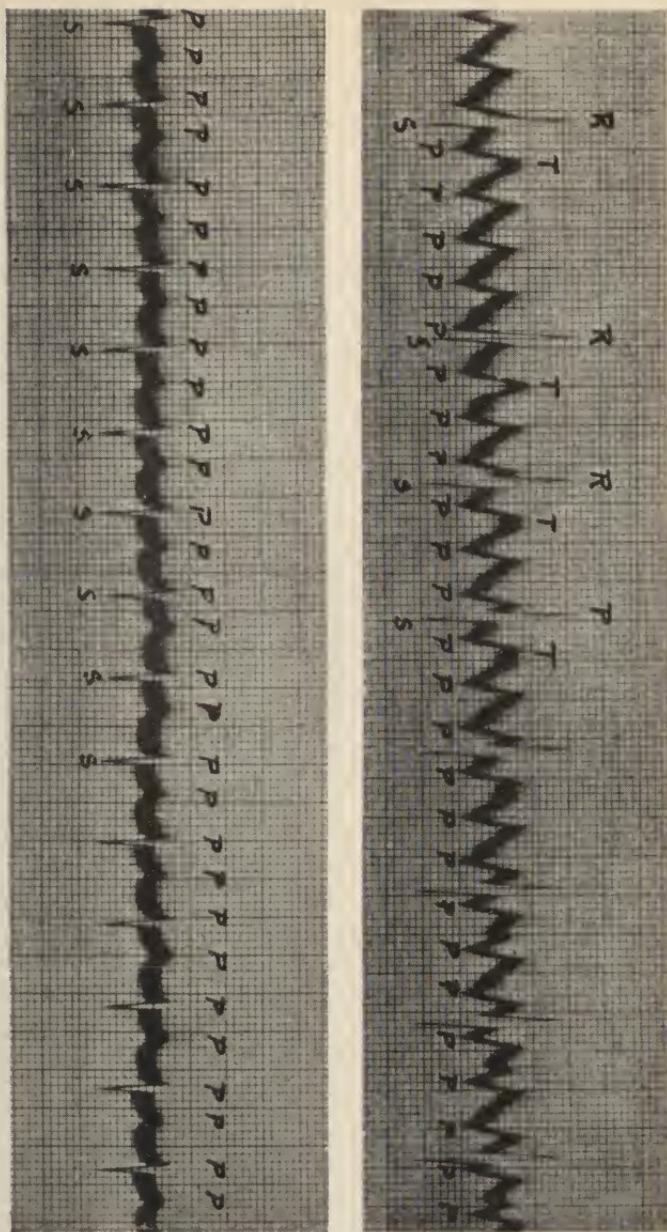


FIG. 21.—Auricular flutter. In the upper electrocardiogram the ventricular response shifts from 5 to 1 to 3 to 1, thus causing an irregularity. In the lower tracing the ventricular rate is regular and just half that of the fluttering auricle.

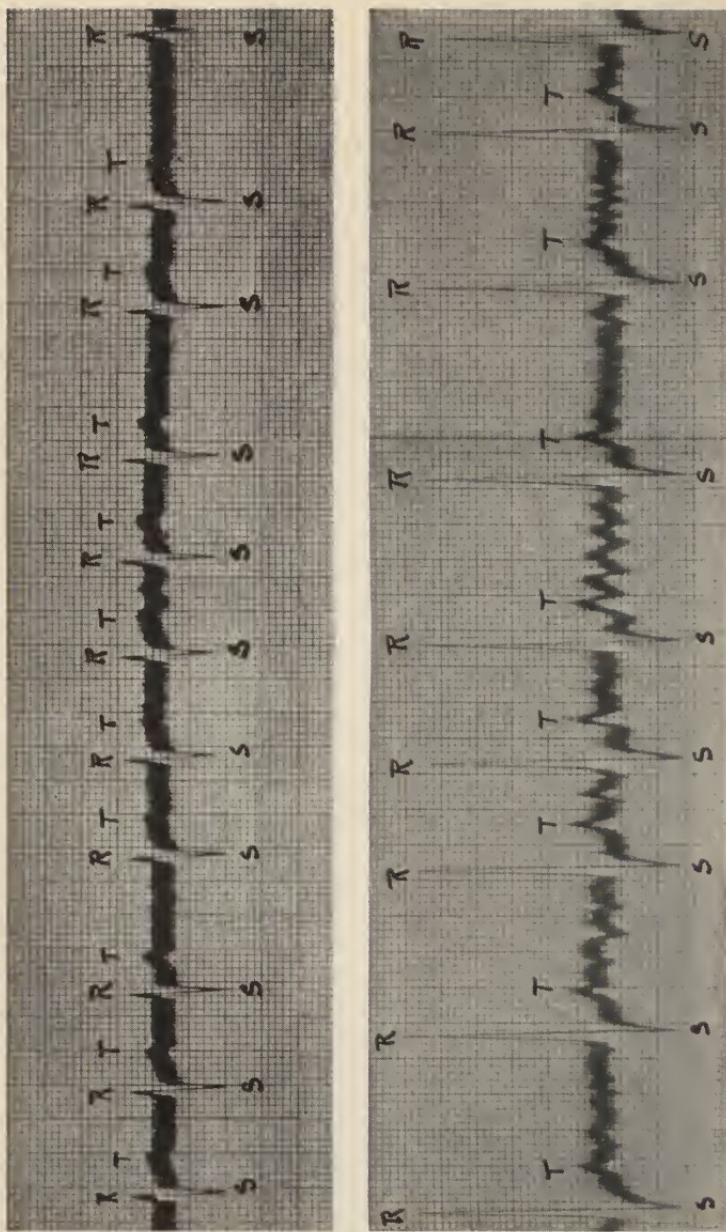


FIG. 22.—Auricular fibrillation. In the lower curve many of the coarse oscillations are shown.

ular musculature that is in process of contraction, but co-ordinate systole of the auricle is in abeyance. It is now known that the mechanism of auricular fibrillation (See Chapter XVII) is essentially the same as that of auricular flutter, but in the former the more rapidly travelling waves follow sinuous paths. The rate of the auricular waves in fibrillation averages 450 per minute.

Auricular fibrillation is readily recognized in the electrocardiogram by the irregular spacing of the R waves and the absence of the normal P waves. The shadow of the string between the successive R-T complexes, namely, during ventricular diastole, varies in the photograph from a flat line to oscillations which may for a few cycles simulate closely the zigzag line seen in flutter. Where the auricular waves are coarse and comparatively regular the condition is termed the flutter-fibrillation type of fibrillation, and by some, impure flutter. It is characteristic of fibrillation that coarse, regular waves,<sup>13</sup> if present, are not so in all of the heart cycles; it is still customary to limit the term auricular flutter to those electrocardiograms showing an absolute and continuous regularity of the auricular complexes.

In typical uncomplicated cases of auricular fibrillation, the ventricular rate is usually well over one hundred and the irregularity is evident, but when some degree of A-V heart-block is present, as when digitalis therapy is in operation, the ventricular rate is lower and may be remarkably regular. Then the absence of normal P waves and the irregular appearance of oscillations during the interven-

<sup>13</sup> They are present in leads taken directly from the chest wall; such leads are more favorable than limb leads for the study of articular activity. *Heart*, viii, 1921.

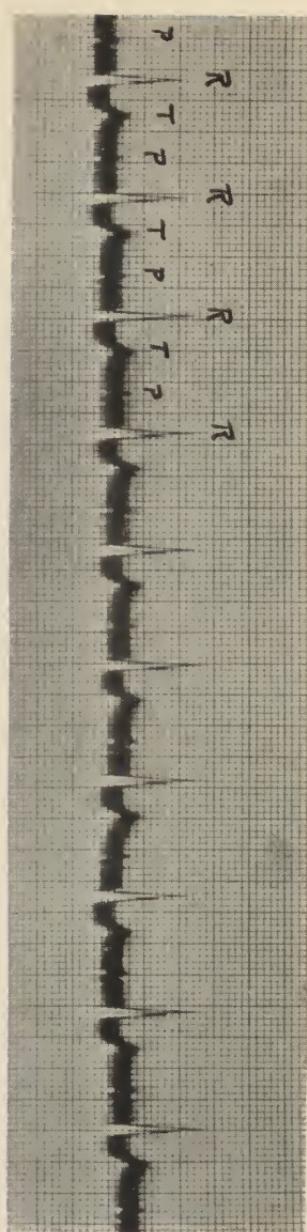
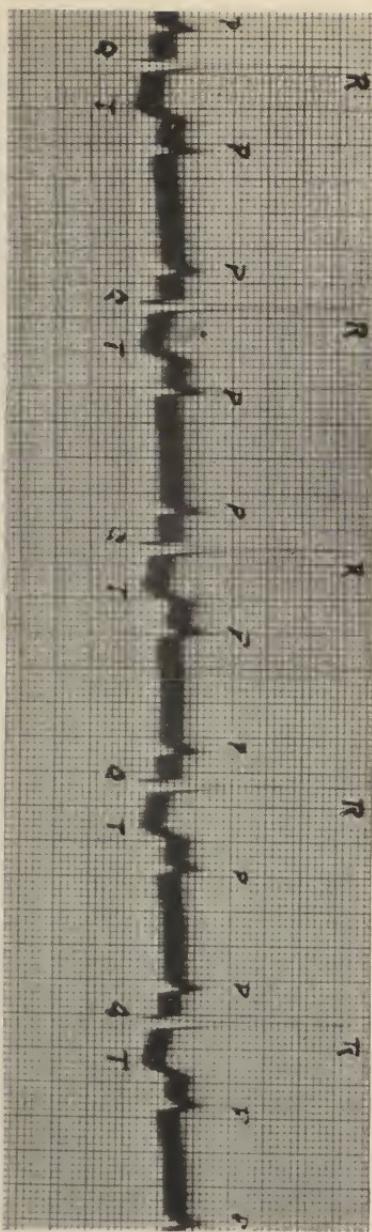
tricular periods of the curve is of distinct assistance in differentiating fibrillation from atrio-ventricular rhythm with P waves buried in the ventricular complexes.

**VENTRICULAR FIBRILLATION.**—This arrhythmia is incompatible with the continuance of the circulation and nearly always results in the prompt death of the patient. As a result it is rarely seen except in records taken just before death. The shadow of the string shows excursions that are abnormal in outline and irregular in spacing. In ordinary clinical work this arrhythmia is unimportant, in the sense that it is not met in the surviving patient.

**HEART-BLOCK.**—This term is applied to defective conduction between the auricles and ventricles. Heart-block may be partial or complete. Partial heart-block is detected in the electrocardiogram by prolongation of the P-R interval, *i.e.*, the latter exceeds 0.21 second. If the P-R interval is as much as one second it is more likely that the ventricle has escaped, or, in other words, did not beat in response to that auricular contraction, but to an impulse originating in the A-V node. In complete heart-block the contractions of the auricles and ventricles are independent of each other. This should be suspected in the electrocardiogram if the P waves appear to have no definite relation to the ventricular complexes, and is confirmed if, on ascertaining their respective rates, the auricular and ventricular waves bear no arithmetical ratio to each other. Thus, if the heart-block is not complete the frequency of the P waves should be to the QRS waves as 1 to 1, 2 to 1, 3 to 1, 4 to 1, etc. An exception to this occurs in partial heart-block when the ventricle fails to reply to certain of the auricular contractions, which are then said to be blocked and result in dropped beats (of the ventricle). A tracing

Fig. 23.—Partial heart-block.

Fig. 23.—Partial heart-block.



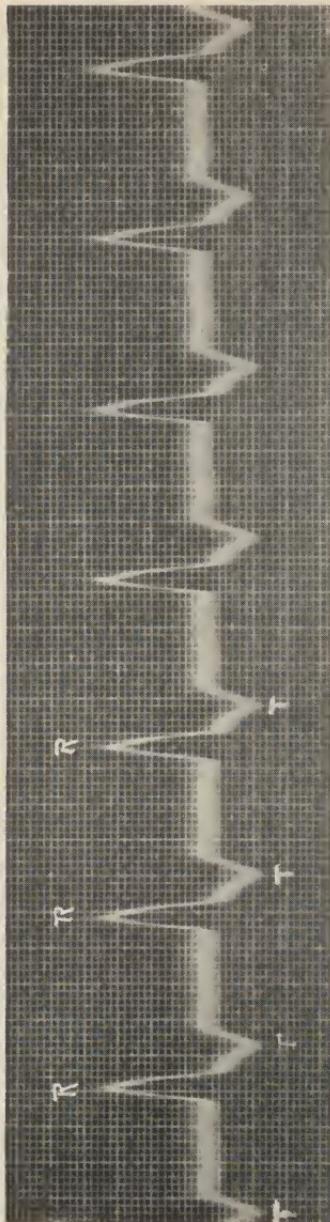
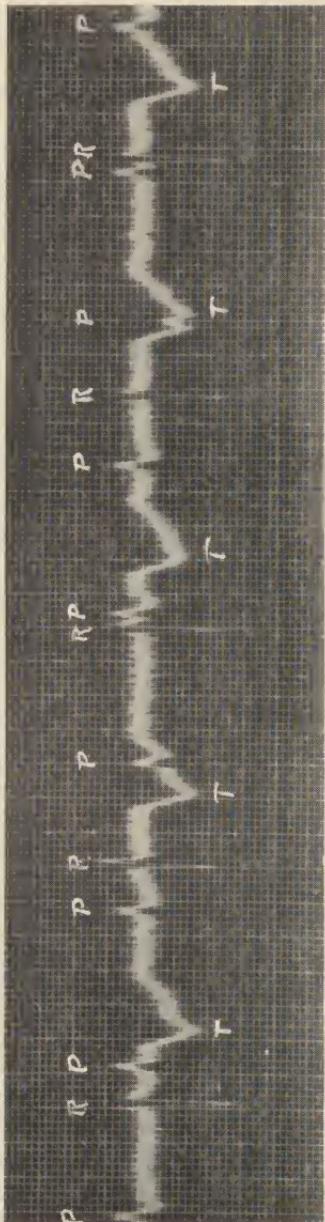


FIG. 24.—Above, Complete auriculo-ventricular heart-block. Auricular rate 79, ventricular 43. Below, Intraventricular block, right bundle branch. In this case, lead I, the P wave happened to be isolectric.

of this condition shows that the ventricle beats in response to many or most of the auricular contractions, and the condition of absolute independence, *i.e.*, complete heart-block, is not, therefore, present. The dropped beats of heart-block differ from sino-auricular block in that the P waves are present and it is only the ventricular complex that is absent. Variation in the P-R interval is common; after the longer pauses there is often a shortening of the P-R interval and then the latter may show a gradual or rapid lengthening, as the case may be, until the P wave is not followed by a ventricular complex, *i.e.*, the P wave is blocked and the ventricular beat dropped.

It has been shown<sup>14</sup> that in heart-block the susceptible region is the junction of the A-V node (node of Tawara) with the auricular tissues or in the node itself. In complete heart-block the ventricle beats in response to impulses originating in its own tissues. The rate of this new rhythm, known as idio-ventricular rhythm, is slow, averaging about thirty per minute. In such cases the level of the block is supposed to be below the A-V node and the new impulse centre below this in the auriculo-ventricular bundle. In some instances, however, complete dissociation is present with a higher ventricular rate, forty to fifty or even ninety<sup>15</sup> per minute. It seems probable that the block is then at a higher level and the impulse centre in the auriculo-ventricular node, which possesses a higher rate of impulse formation than does the conduction bundle on the ventricular side of the node. In either case the ventricular complex appears normal in the electrocardiogram since the ventricle is still

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<sup>14</sup> LEWIS, T., MEAKINS, J. C., and WHITE, P. D.: *Heart*, v: 289, 1913-14.

<sup>15</sup> The Mechanism and Graphic Registration of the Heart Beat, by T. Lewis. 1920. Paul B. Hoeber, New York, p. 306.

responding to supraventricular stimuli, *i.e.*, above the division of the auriculo-ventricular bundle into the right and left branches.

INTRAVENTRICULAR BLOCK, or impaired conduction in the branches of the bundle of His, is indicated if the QRS complex exceeds 0.1 second. Notching and thickening of the wave is also usually present. The T wave, or end deflection, in bundle branch block is opposite in direction from that of the initial wave of the ventricular complex. If the direction of the QRS complex resembles that found in left ventricular preponderance (which see), it is commonly held that the lesion is located in the right bundle branch, and in the left bundle if the waves simulate those of right preponderance.

A block of the right branch is found much more frequently than is one of the left branch. (In fact the latter is very rare.) The usual reason given for this is that the left branch spreads out promptly into a broad sheet and consequently would require a lesion of greater extent than would be necessary to block the branch on the right side, which remains for a space as a comparatively compact bundle. The anatomical studies of Gross,<sup>16</sup> however, suggest another explanation. He finds that the right branch of the A-V bundle invariably receives its blood supply from the left coronary artery by a stout vessel called the *ramus limbi dextri*, which is a terminal artery, while the left branch of the bundle has no specific blood supply of its own, but is nourished by profuse anastomoses of vessels from both sides.

The mistake must not be made of diagnosing prepon-

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<sup>16</sup> GROSS, L.: The Blood Supply of the Heart, in Its Anatomical and Clinical Aspects, New York, Paul B. Hoeber, 1921.

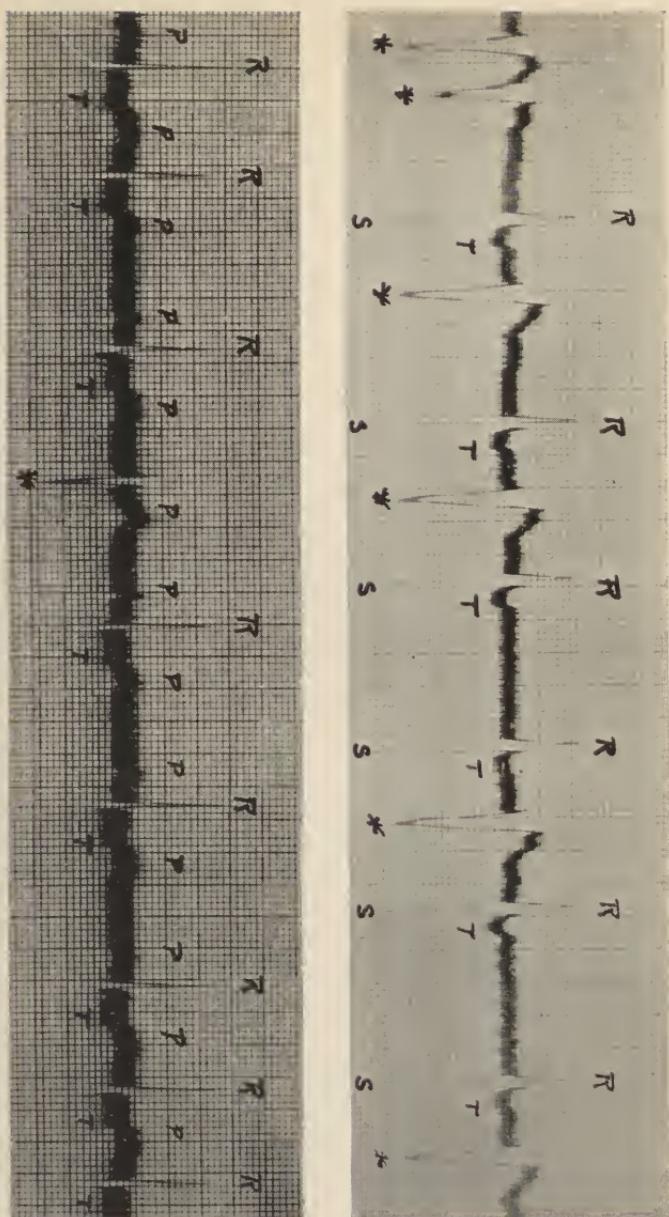


FIG. 25.—Combined arrhythmias.

Above, auricular fibrillation and ventricular extrasystoles causing the so-called bigeminy and trigeminy due to digitalis. Below, partial heart-block interrupted by a ventricular extrasystole. The curve also shows a shifting back and forth of 2 to 1 and 1 to 1 rhythms. The P-R intervals are shorter after the longer pauses. The extrasystoles are marked in both electrocardiograms by an \* placed below. The depression of the T waves in both tracings is due to digitalis.

## CHAPTER V

### THE NORMAL HEART

THE term “normal heart” does not apply to a closely circumscribed standard to which every heart must conform or be classed as abnormal. On the contrary, the heart normally varies, as do other organs, between individuals, and especially in accordance with age, sex, stature, muscular development and manner of living of the individual in whom it is located. If called upon to decide upon the normality of a patient’s heart, the physician can give his opinion with safety only after taking a reasonable history and making a careful examination.

**HISTORY.**—The anamnesis may or may not contain the presence of a disease of etiological significance as regards the heart, and yet of course that organ may have escaped unscathed. Judgment is required in interpreting whether some symptoms which may be described by the patient are due merely to the overtaxing of a normal heart or are the early symptoms of cardiac failure. If, however, there is a history of the progressive increase of the above symptoms during smaller and smaller amounts of physical exertion, grave doubt should exist as to the normality of the heart.

**APEX IMPULSE.**—In most normal hearts, the apex impulse is palpable and frequently visible in the fifth interspace a little within the left midclavicular line, if the patient is sitting or standing, and one interspace higher, if the patient is lying. The true apex of the heart lies slightly (1 cm. perhaps) lower and internal to the outer limit of the palpable impulse. The apex impulse or cardiac im-

pulse frequently extends over both the fourth and fifth interspaces inside the midclavicular line, and in such a case the point of maximum impulse, it should be remembered, is above and internal to the true apex. There is considerable variation, in healthy individuals, in the force of the apex impulse, since the transmission is much influenced by the thickness of the parietes, the width of the intercostal spaces, and the convexity of the chest. Pulmonary emphysema may mask the impulse of a normal heart. The heart may be normal but the apex impulse displaced, if ptosis of the organ is present or if the heart is pushed or pulled from its normal location by outside influences, such as pleural effusion, pneumothorax, pleural adhesions, abdominal tumor, etc.

The apex impulse is usually palpable as a single shock, but it is not uncommon to note the presence of a slight to moderate vibration systolic in time. This vibration, which is sometimes termed a thrill, is most common in young adults, especially when the heart is beating vigorously after exercise or excitement. It should be sharply distinguished from the definite purring thrill which accompanies many cases of mitral disease.

Normally the apex impulse is mobile, with change of position of the patient. If the latter is examined while lying on one side and then on the opposite, the mobility of the apex varies from a half to one and a half inches.

**BORDERS OF CARDIAC DULNESS.**—The outer limit of the percussion dulness of the heart varies somewhat in health. It is particularly dependent, of course, on the position of the heart, which, as demonstrated by radiography, is influenced by the height of the diaphragm, etc. Furthermore, a distinct difference in the location of the

borders of the heart dulness has been shown to result from a difference in the method of percussion employed. Since it is known that "there is no normal heart, but the heart fits the patient," it is more informing to state the relation of the heart border to certain landmarks of the chest. Thus, when using average light percussion and noting the outer limit of slight but distinct dulness, the border of the heart will be found in the fifth interspace, about at the left midclavicular line (nipple line is sufficiently accurate in the male sex); from thence the left border extends upward with a slight inward convexity to the second intercostal space at about 2 to 3 cm. from the sternal margin; and from this point, the percussion outline sweeps downward to a point in the fourth intercostal space, averaging 2 cm. beyond the right edge of the sternum. Below this, the cardiac border usually curves inward to merge with the liver dulness.

The percussion note of dulness will gradually change to that of flatness as the pleximeter finger is moved progressively inward and the examination is continued to the part of the heart between which and the chest wall there is no interposition of lung tissue. This area of the precordia is termed the *superficial cardiac space*, in distinction to the deep cardiac space which encircles it and whose outer border has been detailed above. The limits of the superficial cardiac space, easily determined by the point at which the percussion note changes to flatness, is dependent, obviously, upon the contact of the heart with the chest wall and is much influenced by conditions of increased pulmonary volume, such as emphysema. It is sufficiently accurate to state its outlines in health, as extending from the apex of the heart to the fourth left costal cartilage and

downward in the median line. Because of the influence of the sternum on the transmission of the lung resonance, the right border of the superficial cardiac space, as determined by percussion, is a little to the left of the line where the right lung is in anatomical relation to the heart.

**SUPRACARDIAC DULNESS.**—Percussion at the base of the heart over the great vessels gives varying results in healthy individuals. There may be but slight dulness or a well-marked dull area about 4 to 6 cm. wide, depending upon the proximity of the aorta and pulmonary artery. This area is commonly determined at the level of the second interspace, but it should be remembered that the anatomical position of the beginning of the great vessels is nearer to the third interspace. In drawing conclusions from the results of percussion over the base of the heart, due allowance must be made for the conditions known to influence the relations of the aorta and pulmonary artery to the wall of the chest.

Since the heart and its percussion findings in health are subject to so much variation, it does not seem profitable to attempt to memorize any definite set of figures or landmarks. It may have been noted that the writer has not recommended any one method of percussion as being superior, and it is suggested that the reader continue to employ the method in which he is most skilled. In so doing, experience will gradually demonstrate what data are normal for percussion, as performed by his method and by him. With such a conception of percussion, and especially if the findings are checked at intervals by radiographic examination, percussion, in spite of its limitations, will remain a useful method of cardiac examination.

**HEART SOUNDS.**—The first and second heart sounds

follow in a rhythmic order which is usually sufficiently definite to permit of their differentiation. In comparison with the second sound, the first at the apex is longer, of a lower pitch, louder, and of a booming quality, as opposed to the more clicking or valvular quality of the second sound. At the base, the first sound is usually not longer than the second, and loses much of its booming quality, while in loudness, the second sound takes precedence. Phonocardiography has shown that the first sound at the apex precedes that at the base by 0.06 to 0.07 second. This is explained as due to the fact that the anatomical relations of the heart to the chest wall at the base are such that the initial vibrations of the first sound, which are small, are lost in conduction.

The *first heart sound* may begin with a crescendo tone or acute accent which closely simulates the crescendo murmur of early systole so often present in mitral stenosis. In other instances the sound may appear to have two distinct phases and is described as "split." On acceleration of the heart rate by exercise the apparent splitting disappears.

The *aortic and pulmonic second sounds*, best heard over the second right and second left intercostal spaces, respectively, vary in their relative loudness. In childhood, the pulmonic second sound is louder than the aortic, but after the age of twenty years, the latter gradually becomes the louder of the two. In old age, the aortic second sound in health is nearly always louder than the pulmonic. An individual second sound at the base may be accentuated but not necessarily louder than its fellow. The criterion on which to decide if accentuation is present is if the intensity of sound exceeds that which would be expected in the

heart under examination. The term accentuation is, therefore, applied according to judgment based on experience. It should be emphasized that an accentuated second sound at the base does not necessarily indicate disease; the pulmonic second sound was found to be accentuated in three per cent. of 17,200 recruits at Camp Grant, Illinois, with apparently normal hearts.<sup>1</sup> Some reduplication of the second sounds may be present at the slower cardiac rates.

*Third heart sound.*—A faint low-pitched sound is sometimes audible just after the second sound, from which it is separated by a period of 0.02 to 0.08 second.<sup>2</sup> It is best heard by auscultating just outside the apex, immediately after the patient has assumed the left lateral recumbent posture. Elevation of the limbs (by an assistant) intensifies the sound. If the heart rate is relatively slow, the third sound is more likely to be audible. Thayer<sup>3</sup> reported its presence in 65 per cent. of individuals under forty years of age, but the writer has not succeeded in finding this third sound so frequently.

A systolic murmur at the apex is a feature of many normal hearts. If it is not associated with signs of cardiac enlargement or with the history of etiologic significance as regards cardiac affections, the murmur may often be disregarded. There may also be a systolic murmur at the base, in hearts which are not abnormal. A particularly common form, in children, appears to be due to pressure of the surrounding structures on the infundibulum and pulmonary artery.

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<sup>1</sup> PARDEE, H. E. B.: *Amer. Jour. Med. Sci.*, clviii: 319, 1919.

<sup>2</sup> REID, W. D.: "The Auricular Heart Sounds," *Jour. Amer. Med. Assn.*, 76, 14: 928 (April 2, 1921).

<sup>3</sup> THAYER, W. S.: *Arch. Int. Med.*, 4: 279, 1909.

**BLOOD PRESSURE.**—There is no absolute level, above or below which the arterial blood pressure is abnormal. The height of the blood pressure in health varies according to age, sex, and many physiological conditions. Thus the pressure level may rise ten, twenty, or more points due to the temporary contraction of some of the muscles in the arm, or to excitement, etc. Inequality of the pressure on the two sides of the body, again, may have a physiological explanation. The systolic pressure is usually higher and the diastolic lower in the femoral than in the brachial artery. It must not be thought that the height of the systolic blood pressure, or even the pulse pressure, represent in any accurate manner the actual output of the left ventricle, as there are too many other factors influencing the peripheral pressure. In a rough way it may be said that the systolic pressure should not continuously exceed: 100 to 110, at the age of 10 to 15 years; 115 to 125, at 25 years; 135 to 145, at 40 years; and 145 to 155, at the age of 50 or more. The diastolic averages 60 to 75 at 10 to 15 years, and gradually tends to rise to 85 to 95; it should not exceed 100 at any age.

The **EXERCISE TOLERANCE** should be sufficient to enable the patient to carry on his daily life without untoward symptoms. Experience is the best guide on which to judge how much work a given heart should be expected to do without disturbance. The matter is further discussed in the preceding chapter.

On **RÖNTGEN EXAMINATION** the normal heart varies in size in accordance with the size of the individual in whom it is placed. There are, therefore, no actual dimensions which may be said to be normal. The ratio between the transverse diameter of the heart and the internal diameter of the chest should not exceed 53 per cent.

## CHAPTER VI

### THE CLASSIFICATION OF HEART DISEASE

UNTIL recently the diagnosis of affections of the heart was limited mostly to an estimate of the structural change or pathology that was present. The cause of the disease and the functional condition of the heart are even more important, for upon a knowledge of these latter depends much that is fundamental both in prognosis and treatment. An etiologic diagnosis has, of course, an additional value in the matter of the prevention of heart disease, about which to-day the medical profession is increasingly concerned.

The following is suggested as a tentative classification which emphasizes, according to their relative importance, the etiology, functional condition, and structural change of the various heart affections.

#### I. TYPES OF HEART DISEASE (ETIOLOGIC)

1. Rheumatic heart disease.
2. Septic heart disease. (This is closely related to rheumatic heart disease.)
3. Cardiovascular syphilis.
4. Arteriosclerotic heart disease.
5. Hypertensive heart disease.
6. The heart in hyperthyroidism.
7. The heart in diphtheria.
8. Congenital heart disease.
9. Effort syndrome: irritable heart. This is not true heart disease.
10. Rare conditions. In this group should be consid-

ered cardiac tumors, the heart in obesity, the heart in severe anemia, the beer heart, and perhaps some of the rarer infections, etc.

It is admitted that the causation of some of the above listed types of heart affections is as yet unknown, but it is, nevertheless, believed that an individual case will be more clearly understood and often more intelligently treated if the diagnosis includes the type to which the case belongs. When there exists a wholesome doubt of the etiologic diagnosis, this should be so stated, as such action will serve to stimulate further study of the patient, perhaps with eventual success.

In many instances it is also desirable to state whether the cardiac affection is active or inactive, or to indicate that the point is not yet determined.

## II. THE FUNCTIONAL CONDITIONS

1. *Heart failure.*
  - a. Congestive type.
  - b. Angina pectoris.
2. *The arrhythmias, or disordered heart action.*
  - a. Sinus arrhythmia.
  - b. Sino-auricular block.
  - c. Premature beats (extrasystoles).
    - (1) Auricular.
    - (2) Ventricular.
    - (3) Nodal.
  - d. Paroxysmal tachycardia.
    - (1) Auricular.
    - (2) Ventricular.
    - (3) Nodal.
  - e. Auricular flutter.

- f. Auricular fibrillation.
- g. Heart-block.
  - (1) Auriculo-ventricular.
  - (2) Intraventricular.

- h. Atrio-ventricular rhythm and ventricular escape.
- i. Pulsus alternans.

### 3. *Ability to work.*

This has been classified by the New York Association of Cardiac Clinics as follows:—

- a. Able to carry on the patient's usual activities.
- b. Able to carry on slightly to moderately curtailed activity.
- c. Able to carry on only greatly diminished activity.
- d. Unable to carry on any activity (without distress).

## III. STRUCTURAL LESIONS

### 1. *Endocardial.*

- a. Insufficiency or stenosis (or both) of any one or more of the heart valves.

### 2. *Myocardial.*

- a. Myocarditis (permissible only exceptionally, as part of a clinical diagnosis).
- b. Enlargement. This includes hypertrophy and dilatation.
- c. Ventricular preponderance.
- d. Septal defects (in congenital heart disease).

### 3. *Pericardial.*

- a. Acute fibrinous pericarditis.
- b. Pericardial effusion.
  - (1) Serofibrinous.
  - (2) Purulent.

- (3) Hydropericardium.
  - c. Adhesive pericarditis (including the obliterative form).
  - d. Pneumopericardium and other rare forms.
4. *Position of heart.*
- a. Congenital dextrocardia.
  - b. Acquired dextrocardia.
5. *Great vessels.*
- a. Aorta.
    - (1) Dilatation.
    - (2) Aneurism.
    - (3) Aortitis.
    - (4) Congenital changes (coarctation, transposition with pulmonary artery, etc.).
  - b. Patent ductus arteriosus.

The following examples illustrate how the above classification may be applied in the diagnosis of individual cases.

*Case 1.*—Rheumatic heart disease (inactive), auricular fibrillation (able to carry on only greatly diminished activity), mitral stenosis, and regurgitation.

*Case 2.*—Hypertensive heart disease, pulsus alternans, (unable to carry on any activity), cardiac enlargement.

Study of the above suggested classification will disclose a number of imperfections, and yet the writer feels that its advantages outweigh the limitations. A very similar classification is in use at some of the cardiac clinics of Boston. Some of the rarer conditions are not listed, but in most instances their position in the above classification would be obvious; conditions which are not likely to be diagnosed in life are largely omitted. Only the more important of the conditions represented in the above classification will be discussed in this short treatise.

**SECTION II**

**TYPES OF HEART DISEASE**



## CHAPTER VII

### RHEUMATIC HEART DISEASE

RHEUMATIC heart disease is the term which, in the opinion of the writer, may be advantageously applied to the group of conditions known as; acute or chronic endocarditis, acute pericarditis, mitral stenosis and regurgitation, etc. The primary condition is a rheumatic infection of the heart, and the other terms, it will be seen, are but individual manifestations of the same infection. Such terms as endocarditis and mitral stenosis are open to the objection that they put a false emphasis on the endocardial or valvular changes, whereas Mackenzie has pointed out that, in addition, the myocardium is practically always involved, and on the extent of its impairment depends the future efficiency of the heart.

**ETIOLOGY.**—Rheumatic heart disease was formerly held by some to be bacteria-free, but that it is an infection is now generally conceded; there is less agreement as to the identity of the invading organism. The disease would appear to be a modified septicopyemia.

Rheumatic heart disease is a frequent sequela of acute articular rheumatism, chorea, and tonsillitis. Other conditions whose etiological significance seems established are, scarlet fever, puerperal sepsis, measles, and foci of infection such as abscesses at the apex of the teeth, infections of the nasal sinuses, etc. Rheumatic heart disease, however, may be present even though there is no history of an etiological infection.

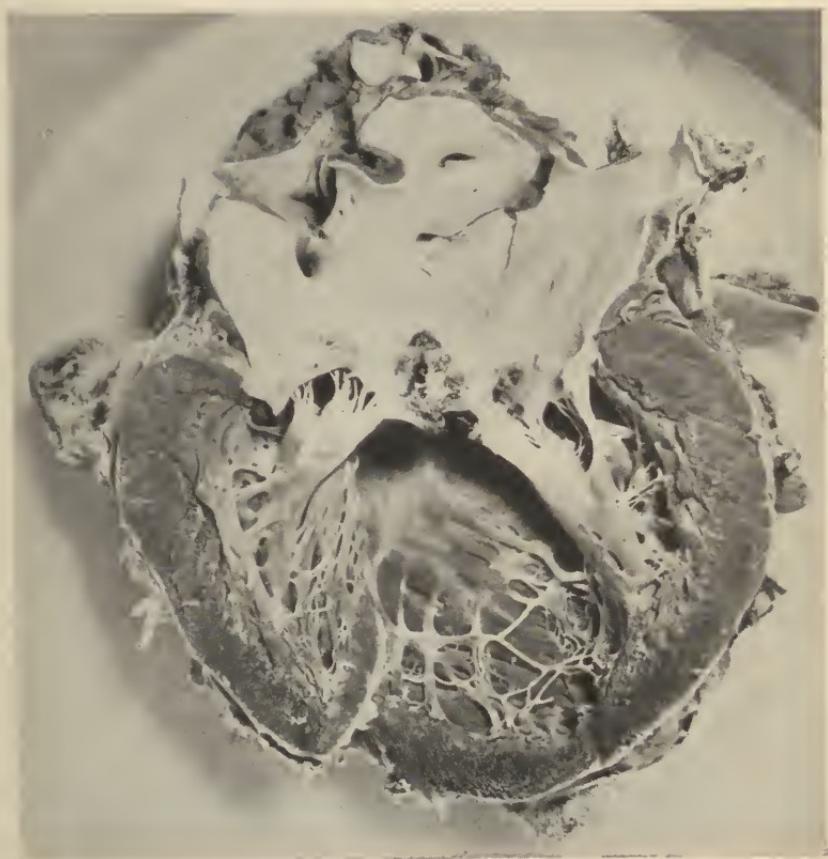
**PATHOLOGY.**—This varies according to the severity and duration of the disease. All three layers of the heart wall are commonly involved, though in the milder cases the lesions of the endocardium may alone be visible. The myocardium may show the so-called Aschoff<sup>1</sup> bodies. These are focal collections of concentrically arranged cells in the neighborhood of minute vessels, or surrounding them. These cells are a little smaller, but otherwise much like those found in Hodgkin's disease. Bacteria are not constantly found in these areas. In the process of healing, the Aschoff bodies are replaced by scars, which tend to produce the condition known as chronic fibrous myocarditis.

The valvular endocardium is usually affected. McCrae<sup>2</sup> found the mitral valve involved in 95 per cent. of the cases, the aortic in 23 per cent., and both valves in 18 per cent. The tricuspid and pulmonary valves and the endocardium adjacent to the valves are less frequently affected. The lesions consist of vegetations or excrescences which are characterized by the terms, wart-like, papular, or even pedunculated. The vegetations are greyish and gelatinous in appearance; they become whiter and firmer with age. The superficial layers are of the nature of blood thrombi, while below are more or less granulating endocardial and subendocardial tissues. Where the lesions are on the edge of the valve, the process may cause a sticking together of adjacent cusps with resulting obstruction. Healing is by the formation of fibrous tissue or scar. Involvement of the chordæ tendineæ may result in a shortening, from the

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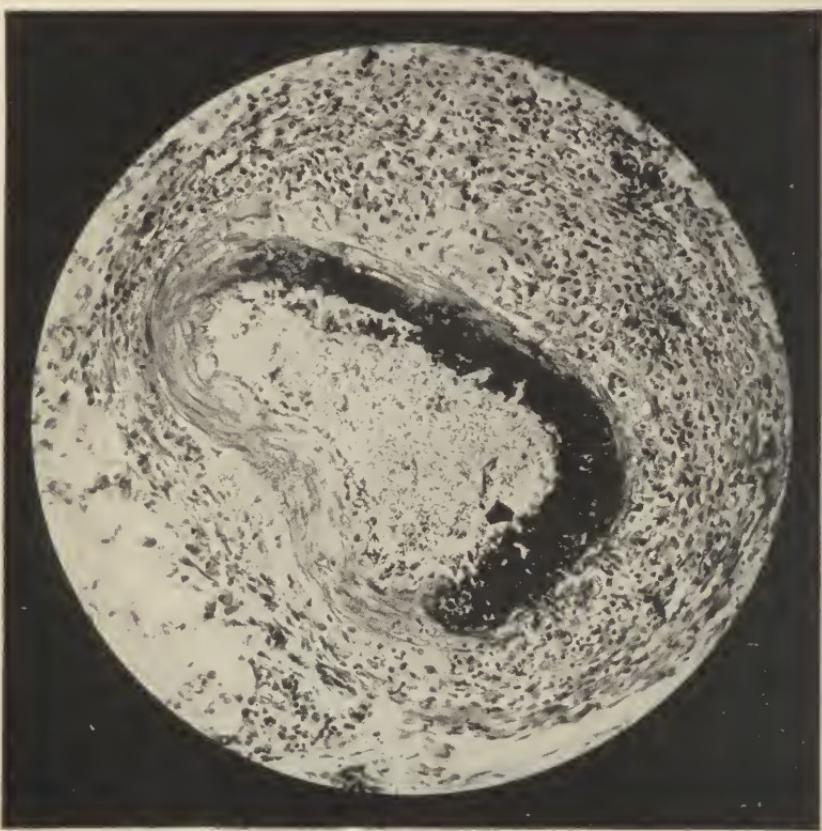
<sup>1</sup> ASCHOFF, L.: *Verh. d. Deut. Path. Gesellsch.*, viii: 46, 1904.

<sup>2</sup> EDWARDS, A. R.: Principles and Practice of Medicine, Lea Brothers and Co., Philadelphia and New York, 1907, p. 195.



Courtesy of Timothy Leary

PLATE 1. Rheumatic heart disease; healed mitral endocarditis with calcification and rupture. Note the thickening and shortening of the chordae tendineae.



Courtesy of Frank B. Mallory

PLATE 2. Rheumatic heart disease; acute infectious lesion of a branch of the coronary artery with surrounding myocarditis.

contraction of scar tissue, and thus impede proper valve closure.

A serous inflammation of the pericardium is common, and the formation of fibrous adhesions between the parietal and visceral pericardium is a frequent sequela.

**SYMPTOMS.**—In rheumatic heart disease there may be practically no symptoms, or those indicating slight to severe cardiac failure may be present. Symptoms, when present, do not directly indicate a rheumatic heart infection, but merely the presence of cardiac failure, the causation of which is to be determined.

The clinical picture will vary considerably in accordance with whether the process is active or inactive. The presence or absence of fever is the most important evidence on which to judge if the rheumatic heart disease is active. In the less acute cases, the fever may be detected only by the keeping of the temperature record over a period of several days. The minimum figure at which fever may be said to be present is, of course, a matter of judgment in an individual case. Perhaps  $99.4^{\circ}$  to  $99.5^{\circ}$  is a reasonable level at or above which fever may be said to be present.

If the disease, held to be the cause, is still present, it is reasonable to consider the cardiac process as active. Following an acute attack, it seems wiser to continue to treat the case as active until fever and symptoms have been absent for some weeks. A progression in the physical signs usually indicates that the infection is still alive in the heart. On this point, however, due allowance must be made for the result of contraction of the damaged tissues after fibrosis has occurred, such as, for example, the appearance of the findings of stenosis of the mitral valve some few months after an acute cardiac infection in which the

## 100 MODERN CONCEPTION OF HEART DISEASE

mitral valve was involved with resulting insufficiency. The belief is now rapidly gaining ground that broken compensation, or to use a preferable term, an attack of heart failure, is nearly always a matter of infection.

Since the term rheumatic heart disease is applied to hearts differing considerably in physical findings and in the associated symptoms, it may make the description clearer to build it around certain clinical types of rheumatic heart disease.

**ACUTE RHEUMATIC HEART DISEASE** occurs preponderatingly in the young. During the course of, or shortly after, an attack of rheumatic fever, or one of the other infections already mentioned as being associated with a rheumatic infection of the heart, examination may show a systolic murmur at the apex with perhaps a weakening of the first heart sound, an acceleration of the rate, and a slight enlargement of the area of dulness. One to several degrees of fever accompanies the above. Since the fever may be due to the primary disease and the cardiac signs indicate merely a weakening of the myocardium resulting from cloudy swelling, it is not possible to be certain at once if a true infection of the heart has taken place. Unless some more positive cardiac sign appears, a decision in favor of rheumatic heart infection is unsafe until there is evidence of persistent enlargement of the heart.

The detection of a diastolic murmur at the aortic area, or along the left border of the sternum, or both, which may or may not mask the second sound, may, on the contrary, be taken as reliable evidence of infection of the heart, as a yielding of the aortic ring from muscular weakness is very rare.

If the heart is auscultated at frequent intervals, a peri-

cardial rub may sometimes be heard. Pericardial friction is usually accompanied by precordial pain, often intense, and not infrequently there will be dyspnoea in addition. In not a few cases, the development of effusion into the pericardium may be observed. It should be remembered that the pericardial friction sound may remain over the base and front of the heart even in the presence of a considerable pericardial effusion, as the fluid tends to gravitate to the posterior and inferior parts of the pericardial cavity. Effusion into the pleural cavity is often associated with the pericardial effusion and occasionally the peritoneum may be involved in addition.

Small nodules about the size of a pea may be palpable in the subcutaneous tissues and along the tendon sheaths of the forearms, especially on the flexor aspect. They appear, as a rule, early in the disease and tend to disappear by resolution within a few weeks after the cessation of the acute infection. Since they are usually painless their presence will not be noted unless a deliberate search is made for them.

Many of the above changes, due to acute rheumatic heart disease, may occur without their presence being noted, as the symptoms are sometimes slight and the heart, therefore, not examined. This very fact is a strong reason why the heart should be repeatedly and carefully examined in any patient suffering from an affection of the "rheumatic group."

There is a less common type of acute rheumatic heart disease, in which the infection is more severe and the process of a fulminating character. It is best considered as an *acute rheumatic carditis*. The patient appears truly ill, and usually suffers from an increasing amount of heart

failure, with a fatal termination in one to three weeks. The fever and pulse rate are high, while the area of cardiac dulness tends to broaden as the heart muscle yields to the massive infection. Both endocardial and pericardial murmurs may be present.

**CHRONIC AND RECURRENT RHEUMATIC HEART DISEASE.**—As a result of a previous acute rheumatic infection of the heart, the latter may present evidence of a healing of the process by fibrosis, or may, from time to time, be subject to further attacks of rheumatic infection. The healed condition may well be considered as being accomplished by a process of scar formation which may be manifest mainly by physical signs and usually by some degree of diminution of the exercise tolerance. The less fortunate will show some or all the evidence of activity described in the earlier part of this chapter. There is considerable analogy to chronic tuberculosis with its residue of abnormal physical signs in the lung, and the tendency of the process to flare up when occasion favors. It has already been stated that it is often difficult to decide if the rheumatic heart infection is active or inactive, and no sharp line can be drawn between the two conditions. For this reason, the remaining description will be based on structural changes in the heart, leaving the decision as to the activity of the infection to be determined in the individual case. The author would again emphasize, that though it may be convenient to describe lesions of an individual anatomical structure, as the endocardium, the rheumatic infection in the same case involves the myocardium, and not infrequently the pericardium, in addition.

**MITRAL REGURGITATION.**—An apical murmur occurring with, and usually following, the first sound, if accompanied with evidence of cardiac enlargement, indicates an

insufficiency of the mitral valve, such as results from damage to the valve from rheumatic infection. A systolic murmur, completely masking the first sound, is generally reliable evidence of organic mitral regurgitation, but it is a good rule to hold that an apical systolic murmur, whatever its transmission or quality, is insufficient evidence of organic mitral regurgitation unless in addition there is evidence of cardiac enlargement of persistent type. There is usually an accentuation of the second sound at the pulmonic area, but this sign is of uncertain value. A history of the occurrence of a disease known to be followed frequently by rheumatic heart disease is helpful, but the latter may exist though the previous history be negative.

**MITRAL STENOSIS.**—A stenotic change of the mitral valve produces a typical picture of rheumatic heart disease. The detection of the signs of mitral stenosis is presumptive evidence of a rheumatic infection of the heart, for though it has been advanced by Allen<sup>3</sup> and others, that syphilis may also cause this change of the valve, it is certainly a rare occurrence and supported by evidence which is questioned by many. To a mild degree mitral stenosis may occur in advanced senile arteriosclerosis. Since the development of fibrosis, and the subsequent contraction thereof, is necessary before the inflammatory process in the endocardium can cause a narrowing of the mitral orifice, it is quite understandable why evidence of mitral stenosis does not appear until several months after the acute infection of the heart. There is a particular value in the diagnosis of mitral stenosis, due to the fact that an infection severe enough to cause eventual obstructive changes in the valve also involves the myocardium to a considerable extent.

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<sup>3</sup> ALLEN, H. B.: *Intercol. Med. Jour. of Australas.*, xiv: 113 (March, 1909).

The myocardial damage associated with mitral stenosis is usually great enough to definitely impair the cardiac efficiency. Twenty-five to fifty per cent. of the cases of auricular fibrillation occur in hearts affected with stenosis of the mitral valve.

Considerable confusion exists in the literature as to the physical signs which may be present in mitral stenosis. For this, and the reasons noted above, the matter will be given a little more attention.

**INSPECTION.**—There is a facies which is suggestive of mitral stenosis. The face appears a little drawn and there is often a flush, sometimes cyanotic, over the cheek bones, with an underlying sallowness, almost a jaundice. This facies occurs usually only in advanced cases. Cyanosis may be present in the lips and ears, or more generally. The patient is often undernourished in type and the hands and feet are frequently cold. Some clubbing of the fingers is common in patients in whom the disease started in childhood.

The *apex impulse*, if visible, is usually in the fifth interspace, at or near the midclavicular line if the mitral stenosis is uncomplicated. If the heart is considerably hypertrophied it may extend nearly to the anterior axillary line, and with extreme preponderant hypertrophy of the right ventricle there may even be a systolic apical retraction in the sixth or seventh interspace well toward or into the axilla. Unusual epigastric pulsation may be found in cases of vertical or "drop" heart (so-called cardioptosis) and of very large forceful hearts, due to any cause, as well as in chronic mitral stenosis, so that the sign cannot be taken as evidence of right ventricular hypertrophy.

*Palpation* over the apex usually locates the impulse in the fifth interspace at or near the left midclavicular line,

but further out and down if the heart is much enlarged. The quality of the impulse varies according to the size, strength, and excitability of the heart, but is rarely heaving or prolonged.

A thrill may accompany the apex impulse. In many cases it may be absent and, when present, varies from a mere vibration to a definite purring thrill. It is of no diagnostic value unless well-marked; then it points strongly to the presence of mitral stenosis. But when the thrill is well-marked and diastolic in time, it will be found that the other evidences of stenosis of the mitral valve are unmistakable. I have never found the thrill of value in a doubtful case of mitral stenosis.

The pulse is not of much assistance in the diagnosis of mitral stenosis. In a typical case it tends to show a pulse pressure of but slight or moderate degree. In rhythm the mitral pulse is not characteristic; absolute arrhythmia is present if auricular fibrillation has ensued as a complication. When aortic regurgitation and mitral stenosis are associated, the latter lesion tends to limit the Corrigan quality of the pulse, *i.e.*, the pulse pressure is lower than in uncomplicated aortic regurgitation.

*Percussion* demonstrates the cardiac apex to be in normal position, or if enlargement of the heart is present, the border of dulness generally extends laterally rather than downward. If the convexity of the curve of cardiac dulness in the left second, third, fourth, and fifth interspaces is outward, the presence of a lesion of the mitral valve becomes increasingly probable. This is, of course, merely evidence of prominence of the left auricle, which is more accurately determined by the Röntgen examination. Increase of the percussion border to the right of the sternum indicates increase in size of the right auricle. Such a con-

dition is not uncommon in mitral stenosis when auricular fibrillation is present, but inasmuch as such enlargement can occur with the auricular fibrillation associated with other types of heart disease, or when a pericardial effusion exists, the finding is not of definite value.

Some impairment of resonance of the apex of the left lung, presumably due to pressure of the dilated left auricle, may sometimes be noted. When this is associated with hemoptysis confusion has arisen as to the presence of pulmonary tuberculosis.

*Nature and cause of the murmur.*—The murmur of mitral stenosis is caused by vibrations in the stream of blood flowing from the left auricle to the left ventricle. If the valve is narrowed to the extent that there is some degree of obstruction to the blood stream, the conditions are right for the formation of a veine fluide or jet, and the vibrations of this are picked up by the surrounding structures, *i.e.*, the valve flaps, chordæ tendineæ, papillary muscles, etc. When these structures vibrate with sufficient intensity, a sound is produced, which, like all sounds, is transmitted in all directions and is subject to refraction as it passes through structures differing in density. The sound, if of sufficient intensity, will, after being subjected to the influences just mentioned, be audible as a murmur in the stethoscope on the chest wall.

Like all heart murmurs, the character of the murmur of mitral stenosis is directly dependent upon the size of the aperture and the velocity of the blood passing through it. The velocity of the auriculo-ventricular blood stream is determined by the difference in pressure in the two cav-

ties. Modern physiology<sup>4</sup> has demonstrated that in early diastole the intraventricular pressure falls to zero but not below it, whereas that obtaining in the left auricle is estimated to reach a maximum of two<sup>5</sup> to fifteen<sup>6</sup> mm. of mercury. When these low pressures are recalled, it becomes quite intelligible why the murmurs of mitral stenosis are so often faint and of a low pitch. Furthermore, studies of the intra-auricular pressure curve show one high peak at the beginning of diastole, when the auricle contains the maximum content of blood, and another at the end of diastole or presystole, at the time of auricular contraction. The work of Yandell Henderson<sup>7</sup> makes it evident that the movement of blood from the left auricle to the left ventricle is greatest immediately after the auriculo-ventricular valves open, and that in hearts beating at a slow rhythm, ventricular filling may be practically complete by the time of auricular contraction. In such cases auricular systole moves but a trifling amount of blood through the valve.

In conformity with the above facts, it is found that the murmur of mitral stenosis is most often audible in diastole, immediately following the second sound. From there it may taper off in a diminuendo fashion, or continue throughout diastole, with, at times, a presystolic accentuation at the time of auricular contraction. The occurrence of a presystolic murmur, not preceded by a murmur earlier in diastole, is a rarity. In the experience of the writer, it is

<sup>4</sup> STARLING, E. H.: Principles of Human Physiology, Lea and Febiger, 1920, p. 946.

<sup>5</sup> PORTER, W. T.: *Jour. Physiol.*, 13: 513, 1892.

<sup>6</sup> BURTON-OPITZ, R.: Text-Book of Physiology, Philadelphia, W. B. Saunders Co., 1920, p. 300.

<sup>7</sup> HENDERSON, YANDELL: *Amer. Jour. Physiol.*, 16: 353, 1906.

common to confuse the true presystolic murmur, due to auricular systole, with the louder crescendo murmur of early systole.<sup>8</sup> The latter murmur is produced by a stream of blood regurgitating through the mitral orifice during the early part of the contraction of the ventricle, and differs from the true presystolic murmur in quality, time, absence of a pause between it and the succeeding sound or murmur, and its occasional persistence when fibrillation of the auricles is present.<sup>9</sup> The crescendo, early systolic murmur, it is true, is often present with mitral stenosis, but it may exist in cases in which the autopsy demonstrates no stenotic change in the valve. It is safer to rely on the murmurs of true diastole. They are usually heard only over a small area near the apex, generally at the point of maximum impulse or internal to it. At times the sound produced by the blood passing through the stenosed mitral valve may be described as a roll or rumble, rather than a murmur.

The first sound at the apex commonly has a quality described as sharp and snappy, and when of this character, should arouse the suspicion of the presence of mitral stenosis. If cardiac failure is present, or if there is a loud systolic murmur of mitral regurgitation masking the first sound, then this sound will not, of course, display the accentuation so often present in mitral stenosis.

The second sound at the base may be faint, or at the apex, absent; but it is usually accentuated over the pulmonic area. As mentioned in an earlier chapter, too much weight should not be put on the diagnostic value of accentuation of the pulmonic second sound.

<sup>8</sup> REID, W. D.: "The First Heart Sound and the Presystolic Murmur," *Jour. Amer. Med. Assn.*, 76: 432 (Feb. 12, 1921).

<sup>9</sup> REID, W. D.: "The So-Called Presystolic Murmur," *Jour. Amer. Med. Assn.*, 77, 21: 1648 (Nov. 19, 1921).

Reduplication of the second sound is common in the mitral stenotic heart; that of the first sound is less frequent. It is possible that some cases of apparent reduplication of the second sound may be due to the occurrence of an abnormally loud protodiastolic third heart sound. In mitral stenosis this third heart sound is frequently very distinct. According to the late Austin Flint, "It is apparently due to the sudden tension on the edges of the mitral valve when, with ventricular diastole, it starts to open widely, but cannot. It may well be compared to the opening of a door protected by a chain latch; and the name 'opening snap' is well chosen."<sup>10</sup>

The *Graham Steell murmur* in cases of mitral stenosis is, in the experience of the writer, relatively uncommon, though some authors have stated that it is often present. Graham Steell, himself, reported it as a rare murmur.<sup>11</sup> It is an early, high-pitched, diastolic murmur, best heard at the third left costal cartilage, and is generally thought to be due to functional regurgitation through the pulmonary valve, due to increased pressure in the pulmonary artery. It is to be distinguished from the murmur of aortic regurgitation, although its time, position, and quality are apparently identical. The distinction rests on the blood pressure and pulse findings, which in the case of aortic regurgitation are usually those of the Corrigan pulse, and on the X-ray and electrocardiographic findings, which are those of right ventricular hypertrophy if the murmur is a Graham Steell. A difference of opinion exists

<sup>10</sup> FLINT, AUSTIN: A Manual of Physical Diagnosis, 8th edition, revised by Henry C. Thacher, Lea and Febiger, 1920, p. 311.

<sup>11</sup> STEELL, GRAHAM: "The Auscultatory Signs, etc., of Mitral Stenosis: A Statistical Enquiry," *Med. Chron.*, Manchester, 1895, iii (N. S.), 409. "Mitral Stenosis," *Internat. Clin.*, 1898, iii (8th series), 1411.

as regards the frequency of this murmur; it seems likely that it is sometimes said to be present when the true condition is aortic regurgitation, associated with mitral stenosis.

The history may disclose embolic infarction to the brain or elsewhere and at times hemoptysis. Both of these may be due solely to mitral stenosis and call for careful examination of the heart. The mechanism of the production of this embolism is quite simple. In the dilated condition of the auricle, often found in mitral stenosis, especially where co-ordinate contraction of the auricle has ceased, as in auricular fibrillation, thrombosis is common. Bits of the thrombus or thrombi are apt to be loosened and directed into the blood stream of the pulmonary circulation in case the embolus comes from a right auricular thrombus, or the systemic circulation (particularly brain and kidneys), if the left auricle is involved. More rarely aphonia, from paralysis of the left recurrent laryngeal nerve, is caused by mitral stenosis.

**X-RAY.**—Röntgen evidence consists mainly in the demonstration of the “mitral shaped heart,” in which the organ appears more rounded than normally. The enlargement is lateral, and particularly in the region of the auricles.

**Electrocardiogram.**—The “typical” tracing may show the auricular or P wave over 3 mm. in height or more than 0.1 second in duration, which is evidence of auricular hypertrophy, and the latter is most commonly found in mitral stenosis. The tracing may also yield evidence of right ventricular preponderance. The absence of both of these changes does not disprove the presence of mitral stenosis, inasmuch as it is only the chronic advanced cases of uncomplicated mitral stenosis that give the “typical” electrocardiogram. In other words, if the electrocardiogram

shows these findings it is fairly certain that mitral stenosis is present; if it does not, it is of no help. Of course, if the auricles are fibrillating the P wave will be absent.

*Aortic regurgitation* may also occur in rheumatic heart disease. Steell<sup>12</sup> found some change, though often trivial, in the aortic valve in 50 per cent. of his cases of mitral stenosis which were subjected to autopsy. Since insufficiency of the aortic valve is not confined to rheumatic heart disease, but is even more common in cardiovascular syphilis, and the physical signs are essentially the same in the two afflictions, the further discussion of this valve lesion will be deferred.

*Aortic stenosis* is an infrequent complication of rheumatic heart disease. It is a relatively rare valvular lesion, and when present the aortic valve is usually insufficient in addition. Though the rheumatic type of heart infection is practically the sole cause of a stenotic change in the aortic valve, for purposes of convenience its further description will be postponed to a later chapter.

*Adhesive pericarditis* may result from organization of the exudate of the acute pericarditis. It is frequently present to some degree without causing signs or symptoms<sup>13</sup> by which it may be diagnosed. If the adhesions extend to the adjacent structures there is more likelihood that abnormal physical signs may result. A systolic retraction of the chest wall at the base of the left axilla and in the region of the eleventh and twelfth ribs in the back

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<sup>12</sup> STEELL, GRAHAM: *Med. Chronicle*, 3: 409, 1895.

<sup>13</sup> In a study of 15 cases at the Massachusetts General Hospital, in which at autopsy the pericardial sac was found obliterated in 7 and united by numerous fibrous adhesions in the remaining 8, the condition was diagnosed in life in but two instances. REID, W. D.: *Boston Med. and Surg. Jour.*, clxxxiii, 18: 386 (Sept. 23, 1920).

(Broadbent's sign), if present, strongly suggests adhesions of the pericardium to the diaphragm. If pleural adhesions complicate, Litten's sign of the excursion of the diaphragm and the respiratory excursion of the base of the lungs, may be abolished. Adhesions to the mediastinum may fix the position of the apex impulse so that it does not show the normal shifting with change of position. At times this tendency of fixation of the heart and adjacent structures may be more clearly appreciated by examination under the fluoroscope.

It should be emphasized that a systolic retraction visible at the apex and between it and the sternum should not be interpreted as due to adhesions of the pericardium. Such a phenomenon has been demonstrated by Mackenzie and others to be produced by hypertrophy of the right ventricle. Diminution or obliteration of the arterial pulse with inspiration (the paradoxical pulse), if present, is not of diagnostic value, since it is present in many normal individuals. Diastolic collapse of the cervical veins, noted by Friedreich, is likewise of uncertain value as an indication of pericardial adhesions, since it is now known to be a feature of the ventricular form of venous pulse, occurring in auricular fibrillation, etc.

Although the heart is frequently of normal size in the presence of adhesive pericarditis, it may be considerably enlarged, and therefore cardiac hypertrophy, out of proportion to or not explained by other lesions, is suggestive of pericardial adhesions. A few cases are seen in which a change from the upright posture is promptly followed by cyanosis and dyspnoea.

In the majority of cases, it is repeated, pericardial adhesions will escape recognition in life. This seems of

less importance since myocardial and endocardial damage are usually present in the same case and a diagnosis of rheumatic heart disease may yet be made.

Various arrhythmias, such as auricular fibrillation and heart-block, and, occasionally, the rarer condition, auricular flutter, may occur in the acute stage of rheumatic heart disease, but are more common when the infection may be said to be old or chronic. Their discussion will be deferred.

**DIAGNOSIS.**—The day has passed when it is sufficient to diagnose any of the structural lesions noted above; today the physician is expected to go further and attempt to determine the underlying cause. Familiarity with the facts already briefly mentioned will usually lead to the correct conclusion. A careful history is often of great assistance. In fact, every patient who is at present ill with or who has previously suffered from any of the diseases mentioned under etiology should be considered as a case of potential rheumatic heart disease until careful examination and the lapse of a sufficient time has proved otherwise. During the acute and subacute stage of the "rheumatic group" of infections, the heart should be examined repeatedly.

Evidence of cardiac enlargement, not otherwise explained, gives strong support for a diagnosis of rheumatic heart infection. Mitral stenosis is practically always of rheumatic origin. The same may be said of stenosis of the aortic valve, but the value of the diagnosis of this lesion is comparatively slight, since it is known that obstructive changes in the aortic cusps are relatively rare, and it is notorious that the clinical diagnosis is frequently unconfirmed at autopsy. Aortic regurgitation may be rheu-

matic but in patients over twenty years of age it is safer to first suspect a syphilitic origin. Pericardial effusion is usually good evidence of rheumatic heart disease. Pericardial adhesions, which cause sufficient signs and symptoms to be diagnosed in life, may likewise be attributed to a rheumatic infection of the heart; tuberculosis also causes adhesive pericarditis, but in the experience of the writer the condition is usually recognized only at autopsy.

Further discussion of the diagnosis of rheumatic heart disease may well be dismissed, after emphasizing that the evidence of its presence should be checked by a complete examination of the patient and the absence of the findings of one of the other types of heart disease.

**PROGNOSIS.**—The prognosis of rheumatic heart disease may be said to be a matter of infection. The severity of the initial attack, the amount of myocardial damage incurred, and the continuance or recurrence of the infection are all important.

If the initial attack is attended with a high fever and marked evidence of cardiac embarrassment, death may result within one to several weeks. The great majority of cases, however, are not ushered in with such severity, and the future depends upon the amount of damage caused by the first attack and especially upon the recurrence of the infection in the heart. If the case is well treated by rest and if success attends the modern method of preventing recurrence by removal of the foci of infection, such as the tonsils, etc., a useful heart may often be retained. The prognosis should, therefore, usually be hopeful but guarded. Some degree of limitation of the exercise tolerance is the rule.

Pericarditis, and even an extensive pericardial effusion, is not necessarily of bad import. It is surprising to see the frequent recovery from even a large effusion into the pericardium.

If mitral regurgitation is the sole evidence of the rheumatic infection, provided sufficient time has elapsed to preclude the appearance of stenosis of the same valve, the heart may well be considered to have suffered but slight damage, for it has been shown that in such hearts the myocardium, the all-important structure, is but little impaired.

Mitral stenosis and aortic regurgitation are much more important, for in these cases it may be assumed that the infection has been of sufficient severity to cause material damage to the myocardium. Auricular fibrillation is a common sequela, especially of the former lesion. Little weight can be placed upon aortic stenosis, as its diagnosis is too unreliable.

Heart-block, auricular fibrillation, and auricular flutter are all serious complications.

**TREATMENT.—*Rest in bed.***—All cases of acute rheumatic heart disease should be put to bed and kept there for weeks or months. How long will depend upon the severity and duration of the acute attack. If fever has been present but a few days and the sole evidence of cardiac infection is the appearance of an apical systolic murmur, it should usually be safe to permit the patient to get out of bed gradually, after the temperature and pulse rate have been normal one to two weeks. A marked elevation of the heart rate, or symptoms of cardiac embarrassment on change of posture, indicate that the heart has not yet sufficiently recovered, and further rest should be prescribed.

Those patients in whom the fever lasted for more than about a week (due allowance, of course, being made for fever which may properly be considered non-cardiac in origin), and in whom the development of the more serious changes, such as aortic insufficiency, pericardial effusion, auricular fibrillation, or heart-block, may have been observed, clearly demand absolute rest for one to several months. It takes time for the heart muscle to undergo the maximum repair for the damage incurred.

Decision as to the need of rest in bed in a case of chronic rheumatic heart disease should be based upon the activity or non-activity of the infection. Absolute rest is desirable for a period long enough to permit healing of the cardiac damage recently acquired. Since many of the physical signs may be due to lesions previously produced and healed by scars, it is usually permissible to let the patient up sooner than if it were known that the heart infection is all of recent origin. The propriety of allowing the patient to cease rest in bed is to be checked as suggested above, in the discussion of the treatment of cases of acute rheumatic heart disease.

In chronic cases and all those in which there is no evidence of present or recent activity of the infection in the heart, rest is not indicated. Restriction of the patient to bed should be prescribed only in case the process in the heart is active or there is a reasonable suspicion thereof (it being recalled that there is a growing belief that there is some degree of infection in most cases of heart failure).

*Removal of the focus of infection.*—A serious attempt should be made in each case to determine, and if possible remove, the focus from which the infection travelled to

the heart. Most frequently tonsillectomy is the needed procedure. This is best done shortly after the acute attack. In some cases, the recurrence of acute attacks may make it unwise to delay further with the removal of the focus, if known.

Something should be said about the attitude of the physician and patient to the operative procedure, tonsillectomy, for example. If the removal of the tonsils is proposed as a cure for rheumatic heart disease, or as an absolutely certain preventive of further cardiac infection, disappointment will frequently result. But if the physician explains that an infection is the cause of the existing trouble in the heart, that the prevention of further infection is all-important, and that the best prospect of accomplishing this lies in the removal of the primary focus, in this case the tonsils, the purpose of and the probability of benefit resulting from the operation should be readily grasped. The tonsillectomy may be offered as the best kind of health insurance.

After the age of forty, the need for tonsillectomy is less certain, as the tonsils are infrequent offenders in the subsequent period of life. The dental infections are then frequent, and it is a good rule to clean out such, even though there is considerable reason to believe that most of the active infection of rheumatic heart disease occurs before the age of forty years.

*Digitalis* is indicated if auricular fibrillation, auricular flutter, or heart failure of the congestive type is present. Since the chief action of this drug is to produce therapeutic heart-block, its administration will rarely prove of value save in cases of accelerated rate of supraventricular origin.

There is considerable doubt as to the propriety of the prescribing of digitalis during the febrile stage. Further discussion of the action and usage of this specific cardiac drug will be postponed to a later section of this book.

*Symptomatic.*—Pain, insomnia, restlessness, dyspnoea, and depression of spirits should be relieved. Attention should be directed to the care of the bowels and the diet. Since the treatment of the above features in rheumatic heart disease is essentially the same as in heart disease of other causation, its details will be deferred to the chapter on treatment.

*Regulation of the patient's life.*—When convalescence is well established, advice should be given as to the amount of exercise that may be proper for the individual patient, and the nature of the employment and daily routine of the patient's life should be discussed. It may be stated at this point that in cases in which the cardiac damage results solely in insufficiency of the mitral valve, or even in uncomplicated aortic regurgitation when there is but slight impairment of the exercise tolerance, but slight restriction should be imposed. In the past it appears that a mistake has been made in limiting too severely the exercise of this type of case. To avoid repetition, the reader is referred to later chapters for further discussion of the above, and on the therapy of pericardial effusion, the arrhythmias, etc.

## CHAPTER VIII

### SEPTIC HEART DISEASE

ALTHOUGH this form of cardiac infection is really a form of rheumatic heart disease, in which the especial virulence of the attacking organism causes the more severe cardiac changes and constitutional symptoms, it seems justifiable, for clinical purposes, to accord it a separate description. It is met in the literature under the terms of malignant, ulcerative, or infectious endocarditis, or, where the causative organism has been identified, the latter is often used as part of the descriptive name of the disease, as, pneumococcic endocarditis, staphylococcic endocarditis, etc. None of these terms seems to the author quite satisfactory. It may be emphasized that the use of the term endocarditis appears objectionable, in that it creates the false impression that the process is limited to the endocardium.

**ETIOLOGY.**—The etiology of septic heart disease differs from that of rheumatic heart disease in that the triad—rheumatic fever, chorea, and tonsillitis—is less prominent. The condition is more likely to be associated with wound sepsis or puerperal infection, or to follow pneumonia, gonorrhea, empyema, etc., but, on the other hand, the history of an etiological disease is often wanting. Previous rheumatic or atheromatous changes in the heart are apt to favor the development of septic endocarditis. The commoner causative organisms are: the streptococci, staphylococci, pneumococci, and gonococci.

**PATHOLOGY.**—The pathology is similar to, but more

severe, than that of rheumatic heart disease. The process involves the myocardium as well as the endocardium, and often the pericardium also. The endocardial changes tend to a rather severe character; vegetations, sometimes of considerable size, form on the valve membranes and in some cases a necrosis of the tissue occurs, leading to an ulceration or perforation of the valve cusps. The vegetations are quite friable and form a ready source of emboli. These emboli are loaded with organisms and thus they act not only in a mechanical way, but also serve as foci of infection where they lodge. The masses which form on the valves and adjacent endocardium are not well channelled with blood vessels, a fact which apparently is of importance in understanding the failure of treatment by means of bactericidal serum.

The spleen is usually enlarged and soft—the septic type of spleen.

**SYMPTOMS.**—The symptoms show considerable variation. There is no typical clinical picture of septic heart disease. The majority of cases may be fitted into one of two groups or types.

The *acute type* begins with violent symptoms and leads to a fatal end in a few days to weeks. Sometimes it may set in with a chill. After one or two days' prodromes, among which headache, fever, and a feeling of being sore all over, predominate, the patient often falls quickly into a stupefied condition, shows high temperature, quick pulse, very rapid respiration, and, on skin and retina, more or less hemorrhages. More rarely, miliary or larger blisters, and, when the staphylococcus is the invading organism, pustules may appear on the skin. Without further local symptoms,



Courtesy of Frank B. Mallory

PLATE 3. Large vegetations on the mitral valve, in septic heart disease due to the pneumococcus.



death ensues in the course of four to seven days, especially if the streptococcus or staphylococcus is the infecting agent.

When septic heart disease complicates lobar pneumonia, as a rule there follow, after a few (three to five) fever-free days, severe febrile general symptoms, which point to the fresh disease, and continue during the ten to twenty days' duration of the affection.

In most cases a cardiac murmur is to be heard. Occasionally the murmur has only a soft blowing character which one not uncommonly hears in other febrile diseases, but more often, it is loud, blowing or scraping. In not a few cases the murmur is entirely lacking, as in many cases the soft vegetations hinder neither the normal flow of blood nor the regular closing of the valve.

As a result of emboli, especially in patients in whom the pneumococcus is the attacking organism, a panophthalmia may occasionally occur and still more rarely a purulent meningitis. Other disturbances resulting from emboli are abscesses in the liver, spleen, and bowel. As a result of an embolus lodging in the mesenteric artery, fatal intestinal hemorrhage has occurred. A renal abscess may develop, especially in the staphylococcic infection. The organism can be identified in the urine.

The *chronic type* begins much less severely. Occasionally there is a sore throat, influenza, or a severe cold with a fever preceding, from which a proper recovery has not ensued. The patients feel weak, often have pains or twinges in the extremities, and have occasional light chills. In other cases these symptoms have occurred without the patient being aware of any preceding acute illness. The temperature is elevated,  $100^{\circ}$  to  $102^{\circ}$  being the average.

The picture may remain unchanged for a whole month. Occasionally chills are entirely absent, but in their stead are sweats, especially at night or in the early morning.

In other cases, lasting over five or six months, in which the ordinary streptococcus is at work, periods of intermittent fever slip in; more rarely, days with a mild elevation of temperature and, at irregular intervals, violent chills occur. The cutaneous and retinal hemorrhages and spots in the skin are apt to appear, not all at once, but in successive crops.

Energetic, work-loving natures lose their strength and take to bed. On examination at this stage there will commonly be found characteristic heart murmurs, enlargement of the liver, and swelling of the spleen. Most patients of this class experience more or less active rheumatic pains, which affect the joints less severely than the adjacent muscular tissue or the periosteum of the long bones. In some instances there may be sharply circumscribed painful areas at the tips of the fingers.

Fleeting effusion into the joints may take place. Also there follows, towards the end of life, a severe nephritis, in which numerous colorless red corpuscles and often masses of epithelial casts appear. Not uncommonly the picture is that of a severe hemorrhagic nephritis.

In contradistinction to the acute form, chronic septic heart disease only exceptionally leads to purulent metastases. Renal and splenic infarcts are common and often without symptoms. Embolic processes in the brain may produce aphasia, hemiplegia, or convulsions, but these are often of a temporary nature. Gangrene of an extremity

and aneurisms are further results of damage wrought by emboli.

**DIAGNOSIS.**—The diagnosis offers considerable difficulty. Septic infection of the heart should be suspected where there is a fever of protracted duration of an irregular type and associated with chills or sweats. Evidence of emboli, petechiæ in the skin, a palpable enlargement of the spleen, sallowness of the complexion, some degree of anemia, and any of the symptoms mentioned above, point strongly to the presence of a septic infection of the heart. The diagnosis is confirmed by the finding of numerous colonies of the infecting organism in repeated cultures of the blood. Repeated blood cultures may, however, remain sterile.

Other diseases which may at times be simulated are: typhoid fever, miliary tuberculosis, malaria, other cardiac diseases, pneumonia, meningitis, results of emboli, uremia, pyelitis, diseases of the biliary tract, and acute articular rheumatism. Limitation of space forbids the discussion of all these affections.

*Typhoid fever* and the *miliary form of tuberculosis* offer the greatest problem in differential diagnosis. A careful history and study of the symptoms and physical signs are essential. The laboratory findings are often the most successful means of differentiating these diseases. The white count doesn't help much, as a leucopenia may occur in septic heart disease as well as in the other two conditions. A positive Widal reaction (unless the patient has had a course of the typhoid vaccines, as did the soldiers in the recent war) and the finding of typhoid bacilli in the blood culture, will establish the presence of typhoid

fever. Sputum and lumbar punctures may sometimes assist in the diagnosis of miliary tuberculosis. Blood cultures which are positive for organisms other than the typhoid bacillus prove the diagnosis of septic heart disease. It is suggested that the blood culture should show numerous colonies and that the finding should be repeated in one or more subsequent blood cultures, in order to rule out mistakes due to contamination, and errors in laboratory technique.

*Septicemia*, it is true, may yield a positive blood culture. The presence of a septic wound, or evidence of puerperal sepsis, and the relative lack of the findings of cardiac involvement, are generally sufficient to establish the diagnosis of septicemia in preference to septic heart disease, a similar but more localized affection.

**PROGNOSIS.**—The prognosis is almost always fatal. The patient gradually loses ground, with more and more anemia and often with increasing damage from the discharge of septic emboli into the blood stream. Recoveries exceptionally occur.

**TREATMENT.**—The treatment. The patient should be at rest in bed. Fresh air and good nursing are important. The diet should be generous; the aim being to aid the patient in his fight against the infection by supplying plenty of nourishing food. Attention should be directed to the bowels and symptoms treated as they arise.

Billings reports some success from the use of cacodylate of soda in a dosage of gm. 0.6 to 1.0 (gr. 10 to 15) daily hypodermically, and the same amount every two to three days intravenously. Others have used small doses of arsphenamine.

It would seem that some sort of specific therapy should offer a logical method of attack, but experience has shown that it is of but little avail. Vaccines are not indicated. The free use of bactericidal serum from a horse made highly immune to the organism obtained from the blood culture of the patient has been tried. A parent or near relative has been made immune in a similar fashion and then used as a donor for repeated blood transfusions.<sup>1</sup> As a result the organisms have disappeared from the patient's blood for about twelve hours and then return in large numbers.<sup>2</sup> A probable reason is that, due to the lack of blood vessels in the septic focus in the heart, there is a failure of penetration of the bactericidal serum and the masses of organisms in the depths of the lesion remain unscathed.

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<sup>1</sup> LEVISON, L. A.: *Jour. Lab. and Clin. Med.*, St. Louis, 4: 191 (Jan., 1921).

<sup>2</sup> Personal conversation with Benjamin White, Director of the Biologic Laboratories of the Massachusetts Board of Health.

## CHAPTER IX

### CARDIOVASCULAR SYPHILIS

INFECTION of the heart and the aorta by the spirochete of syphilis produces one of the most important types of heart disease. Statistics<sup>1</sup> from various clinics show the presence of cardiovascular syphilis in 3.5 to 7 per cent. of the total autopsies and in 75 to 85 per cent. of the autopsies on bodies known to have been infected with syphilis. In 40 of the 54 cases in which this disease was found at post-mortem examination at the Massachusetts General Hospital, the condition was held to be the primary cause of death. In three of the fourteen remaining cases, in which the cardiovascular syphilis was listed as a secondary cause of death, the process was advanced to a serious degree.

ETIOLOGY.—As early as 1724, Lancisi,<sup>2</sup> according to Allbutt, associated “Aneurysma Gallicum” with “impure coitus” signs of syphilis elsewhere in the body, but it has been only since the discovery of the spirochete in the aortic wall in 1906 and 1907 by Reuta,<sup>3</sup> Benda,<sup>4</sup> and Schmorl,<sup>5</sup> and in the myocardium by Warthin<sup>6</sup> in 1916, that the true nature of cardiovascular syphilis has been understood. The lesions are, then, due to the invasion by the spirochete of the tissues of the heart and aorta.

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<sup>1</sup> REID, W. D.: *Boston Med. and Surg. Jour.*, clxxxiii, No. 3: 67 and 4: 105 (July 15 and 22, 1920).

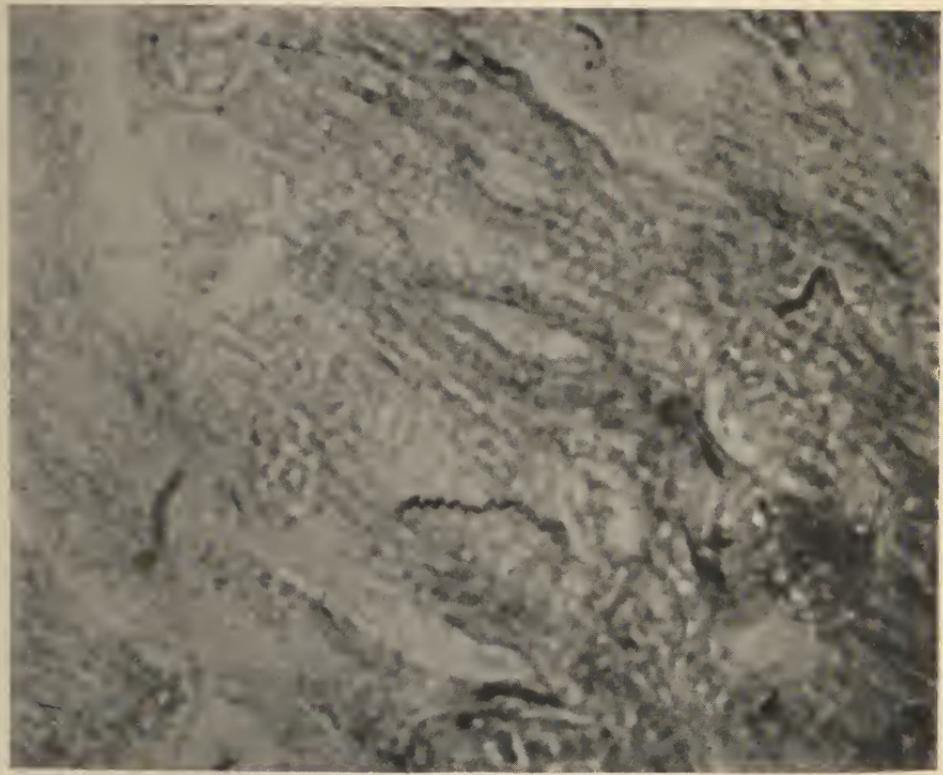
<sup>2</sup> LANCISI: “*De novissime observatis abscessibus,*” c, xviii, ed. 1724.

<sup>3</sup> REUTA: *Munch. med. Woch.*, liii: 778, 1906.

<sup>4</sup> BENDA, C.: *Berlin. klin. Woch.*, xlivi: 989, 1906.

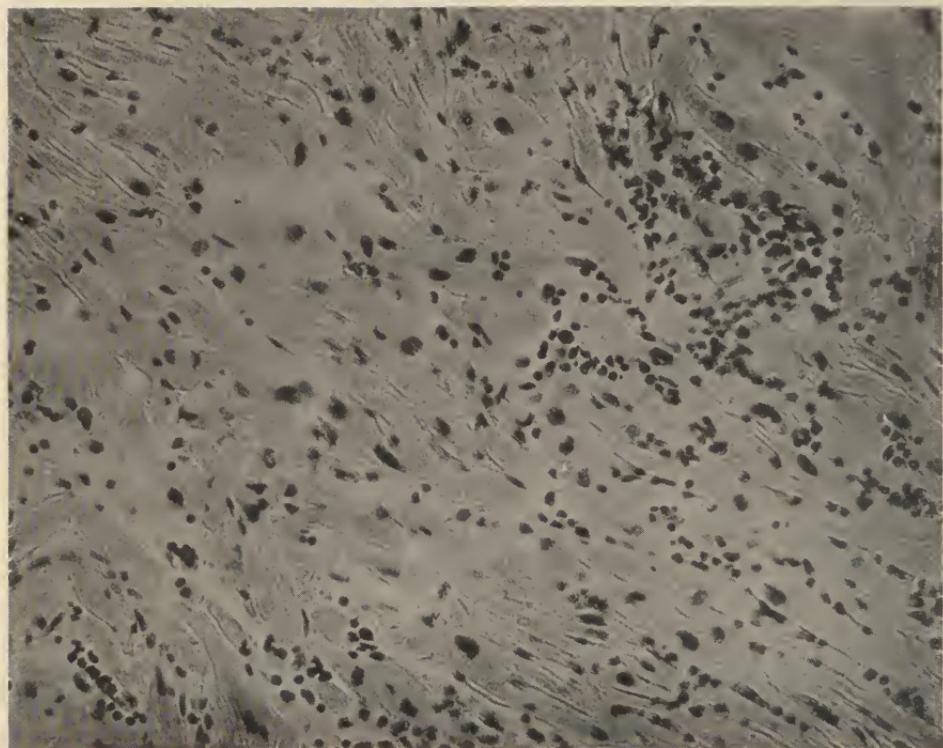
<sup>5</sup> SCHMORL: *Munch. med. Woch.*, liv: 188, 1907.

<sup>6</sup> WARTHIN, A. S.: *Amer. Jour. Med. Sci.*, cliii: 508, 1916.



(Courtesy of A. S. Warthin.)

FIG. 26.—Spirochetes in the myocardium.



Courtesy of A. S. Warthin.)

FIG. 27.—Syphilitic myocarditis.

The involvement of the heart and aorta may begin before or during the so-called secondary stage of syphilis. However, sixteen years was the average period elapsing between the primary lesion of the luetic infection and the appearance of symptoms of circulatory disease, in the cases treated at the Massachusetts General Hospital;<sup>7</sup> the shortest was six months and the longest thirty-three years.

Men are attacked about three times as frequently as women. The age period from 35 to 50 years shows the highest number of cases. Congenital cases are not infrequent at the age of 15 to 20 years<sup>8</sup> and are well known in syphilitic infants. Laborious or athletic pursuits appear to be important determinants. Alcohol is probably unimportant, as the disease occurs in total abstainers.

**PATHOLOGY.**—In recent years there has been an appreciation of the fact that, coincident with the syphilitic lesion in the aorta, the heart proper is usually involved. To the naked eye, the heart may show dilatation, hypertrophy, atrophy, and patches of fibrosis in the wall of the left ventricle. In congenital cases the wall of the right ventricle may be chiefly affected.<sup>9</sup>

In many cases the fibroid changes are detected only on microscopic examination. To follow Warthin's description,<sup>9</sup> the essential lesion is an interstitial myocarditis, characterized by infiltration with lymphocytes and plasma cells along the vessels between the muscle fibres. The entire heart wall, from epicardium to endocardium, including the papillary muscles, may be involved in the infiltration.

<sup>7</sup> REID, W. D.: *Jour. Amer. Med. Assn.*, lxxiii: 1832, 1919.

<sup>8</sup> ALIBUTT, C. A.: Diseases of the Arteries, including Angina Pectoris, Macmillan & Co., 1915, ii: 140.

<sup>9</sup> WARTHIN, A. S.: *Amer. Jour. Syphilis*, 2: 425 (July, 1918).

tions; but in the average case they lie nearer to the endocardium. In the great majority there are areas of healing by fibrosis associated with areas in which the process is active. A progressive fibrosis of the heart muscle eventually takes place in all cases.

Sclerosis of the larger branches of the coronary arteries is rare, even in cases affected by angina pectoris. In the most severe cases there is infiltration around the smaller arteries. An aneurism of the wall of the left ventricle is not uncommon. True gummata in the myocardium are relatively rare.

The aortic valves showed slight to moderate fibrous thickening in about two thirds of the Massachusetts General Hospital cases, while in another ten per cent. the damage was more extensive. The correlation of the clinical and the post-mortem evidence<sup>10</sup> indicates that the insufficiency of the aortic valve, present in 40 per cent. of the cases, is more often dependent upon a yielding of the aortic ring than upon actual damage to the valve curtains. Aortic stenosis is a great rarity. Syphilitic changes of the mitral and tricuspid valves apparently do not occur.<sup>9, 11</sup>

There is much variation in the extent of the pathological changes in the arterial system. The lesions are mostly found in the wall of the aorta and predominatingly in the ascending and transverse arch. This aspect of cardiovascular syphilis has been so evident that the disease is commonly referred to in the literature as syphilitic aortitis. In 94 per cent. of the group of cases examined at the Massachusetts General Hospital<sup>10</sup> the ascending portion

<sup>10</sup> REID, W. D.: *Boston Med. and Surg. Jour.*, clxxxiii, No. 3: 67 and 4: 105 (July 15 and 22, 1920).

<sup>11</sup> BROOKS, H.: *Amer. Jour. Syphilis*, 5, 2: 217 (April, 1921).

of the arch was affected, but not infrequently the process was as extensive in the transverse arch, and sometimes more so.

In its acuter stages syphilitic infection of the aorta is not arterio-sclerotic but inflammatory; it is, strictly speaking, aortitis. The primary site is along the vasa vasorum in the adventitia; the media is soon involved and the intima last of all. Around the vasa vasorum are found collections of plasma and lymphoid cells, and, in the more acute foci, the treponemata may be demonstrated by the Levaditi silver impregnation method. A definite gumma is rare. There may be large areas of healing by scar. It is the irregular distribution of the fibrosis and the necrosis, *i.e.*, the reparative and the destructive changes, which gives the irregular puckered appearance to the aortic wall in cases of advanced aortitis. The absence of calcification in the luetic lesion is a contrast with its prevalence in arteriosclerosis. Warthin<sup>12</sup> emphasizes that the pathological diagnosis of syphilis is essentially microscopic, as to the naked eye the tissue may appear sound.

When the process has caused the aorta to dilate considerably it is often a matter of personal choice with the examiner whether he employs the expression syphilitic aortitis with dilatation or syphilitic aortitis with aneurism; there is no hard and fast line between the two conditions. Large aneurisms of the saccular or dissecting types may occur. These big aneurisms involve the adjacent structures by pressure, and by a process of erosion may penetrate and finally rupture with fatal hemorrhage into the pericardial sac, the chest wall, trachea, esophagus, etc. A

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<sup>12</sup> WARTHIN, A. S.: *Amer. Jour. Syphilis*, 2: 425 (July, 1918).

great majority of aneurisms which involve the aorta before it pierces the diaphragm are due to syphilis, but rarely, usually of the small saccular type, they may result from non-luetic infections.

**SYMPTOMS.**—Cardiovascular syphilis is for the most part insidious. Such symptoms as occur are mainly due to the cardiac lesions rather than to those of the aorta, save for the symptoms caused by pressure when an aneurism has formed. It does not seem practical to classify the cases in relation to the anatomical lesion, but perhaps they may better be divided into acute or chronic according to the intensity of the symptoms. Tides of activity occur.

If there be symptoms, pain is prominent. It varies from a sense of tightness or burning about the upper sternum to the utter torture of severe angina. It may come in attacks, be associated with exertion, or be present almost continuously. In location and radiation the pain of cardiovascular syphilis is wont to resemble that of angina pectoris (which see).

Shortness of breath is common. It may be associated with rapid heart action and other evidence of cardiac embarrassment. Cough and weakness are further symptoms.

Fever of a low-grade "septic" or irregular type may occur, but is of little diagnostic value, as it is rare that careful examination of the patient fails to show the presence of some other condition which might cause the rise in temperature. Also, of course, fever may be present at various times in syphilis, but does not *per se* indicate invasion of the circulatory tissues.

In cases in which the luetic infection has seriously impaired the integrity of the heart and aorta, symptoms

consistent with advanced heart failure will be in evidence. When an aneurism is present pressure symptoms may arise. A rather strident whistling respiration on exertion, while by no means an initial sign, may yet long precede other respiratory signs as a symptom of pressure. A "brassy cough" and partial aphonia are frequent results of involvement of the left recurrent laryngeal nerve in its course round the arch of the aorta. The direction in which pressure is exerted by an aneurism and the rapidity with which dilation occurs and grows are undoubtedly determining factors in the production and absence of symptoms.

**PHYSICAL SIGNS.**—The evidence of luetic infection of the circulation that is obtained by physical examination varies according to the nature and the extent of the lesions present. In many cases physical signs may be essentially absent. The discussion of the physical signs may be considered to best advantage, perhaps, under inspection, palpation, percussion, and auscultation.

*Inspection* is of value only in advanced cases. Thus, a visible throbbing of the carotid suggests insufficiency of the aortic valve, which, in most cases in adults, is known to be of syphilitic origin. A visible pulsation in the suprasternal notch may mean a dilated aortic arch. Again, any visible pulsation in the second or third intercostal space suggests aneurism. Enlargement of the cervical veins may be noted, but should not be expected save with cases complicated by aneurism and with other signs of pressure. Inequality of the pupils from irritation of the cervical sympathetic nerve may occur.

*Palpation.* A palpable impulse in the suprasternal notch, due to dilation of the arch of the aorta, is, at best,

not a particularly reliable sign, as it is not always present and also occurs in arteriosclerosis and in some other non-specific conditions. In case aortic regurgitation is present, the apex impulse is usually moved downward and to the left and accompanied by the Corrigan or collapsing type of pulse. An expansile pulsation, lateral to the base of the sternum, is strong evidence of aneurism.

*Percussion* may be useful to detect enlargement of the first part of the aorta. The suprachardiac dulness is most marked in the midmanubrial region, and commonly the dulness is greater to the right than to the left. In rare instances an area of dulness has been detected to the left of the third and fourth dorsal vertebræ due to a dilation or aneurism of the adjacent aorta.

*Auscultation*.—The second sound at the second right costal cartilage often is of a duller note than normal, but practically the same quality may occur in arteriosclerosis. A systolic murmur, often very faint and soft, but sometimes loud and rough, at the aortic area is frequently present in cardiovascular syphilis, and occasionally is accompanied by a thrill, not necessarily indicating a stenotic change in the aortic orifice. If the luetic infection has caused an insufficiency of the aortic valve, the characteristic diastolic murmur of aortic regurgitation will be noted. This, of course, may or may not mask the second sound. At the apex a systolic murmur due to an insufficiency of the mitral valve, and of muscular origin, is commonly present in addition to the aortic findings. And in some cases, the Austin Flint murmur at the apex may be a further finding. A friction rub over the base has been reported.

An abnormal rhythm may control the heart. Partial

or complete heart-block, resulting from myocardial lesions, is particularly prone to occur. Premature beats are common. Less frequently, auricular fibrillation or other arrhythmia may obtain.

There is nothing characteristic in the *blood pressure* readings in cardiovascular syphilis; it remains essentially normal unless aortic regurgitation or some condition (not syphilitic) causing hypertension complicates the case.

As in other forms of syphilis, the Wassermann reaction has been found to vary from strongly positive to negative. There were 7, or 25 per cent., full negative results in 27 Wassermann tests performed at the Massachusetts General Hospital on cases proven shortly after, at autopsy, to have definite cardiovascular syphilis.

**RÖNTGEN FINDINGS.**—It is inconsistent with our knowledge of the pathology of cardiovascular syphilis to expect that evidence of its presence can be obtained in early cases by radiographic examination. But in the more advanced cases, particularly when the aorta has undergone alteration in size and contour, the X-ray often produces very definite findings.

A marked prominence of the aortic shadow to the right, just above that of the right auricle, is almost always due to syphilitic aortitis. A general enlargement of the shadow of the aorta may occur, but must be distinguished from the result of changes in the aortic curve, occurring in some other conditions. An aneurism, if present, is usually distinguished on radiographic examination. The value of this is better appreciated when it is remembered that in one half the cases of the saccular type, there are no sounds or murmurs over the aneurism.

If the width of the great vessels, obtained by Röntgen mensuration, is more than one half that of the heart, syphilitic invasion of the aorta is almost certainly present. In doubtful cases, the mensuration should be repeated in a few weeks or months, to note if the aorta is increasing in size. The heart often shows enlargement more markedly in its long diameter, and especially in those cases in which insufficiency of the aortic valve is present.

**DIAGNOSIS.**—A clear distinction must be made between “syphilis of the heart” and “the heart in syphilis,” as it seems established that the heart is not involved in every patient suffering from syphilis. On the other hand, it is the part of wisdom to consider every patient known to have syphilis as a potential case of cardiovascular syphilis.

Syphilis should be considered in any case of cardiovascular degeneration of obscure origin. Every case of aortic insufficiency, or heart-block, developing in an adult not giving a clear history of rheumatic infection, should at once arouse the suspicion of specific disease. A definite history of syphilitic infection and a positive Wassermann reaction are of considerable value, but they may be absent. The observation of syphilitic skin lesions, or some other mark of lues, may suffice to turn a possible into a probable diagnosis of cardiovascular syphilis. The X-ray, in all but very early cases, usually gives the most definite confirmatory findings.

Though easy to diagnose in a typical and advanced case, cardiovascular syphilis is almost impossible to diagnose in the early or latent type. There is no one point on which a diagnosis is to be made, but each suspected case requires careful attention to the history, especially to the symptoms

and physical findings, and is to be checked up by radiographic examination and Wassermann test. It is only after all the facts have been collected that definite conclusions should be drawn, and at times only a tentative diagnosis is possible. In this position the diagnosis may remain until the results of the therapeutic test for syphilis have been observed. This consists preferably of four to six injections of arsphenamine at weekly intervals, followed by a course of seven to eight intramuscular injections of a mercurial salt. Of less value, but for certain reasons sometimes employed, is the oral administration of hydrarg. cum creta 0.06 to 0.12 gm. (gr. i to ii) *t.i.d.*, with 0.3 to 1.2 gm. (gr. 5 to 20) of potassium iodide *t.i.d.*, for about six weeks.

**DIFFERENTIAL DIAGNOSIS.**—*Non-syphilitic aortitis* occurs<sup>13</sup> in rheumatic fever, influenza, septic heart disease, anthrax, erysipelas, small-pox, typhoid fever, diphtheria, measles, scarlet fever, pneumonia, malaria, tuberculosis, and gonorrhea. It is relatively rare, often symptomless, and usually overlooked. Even small aneurisms or perforations may occur. Bennert<sup>14</sup> has established a rule that aneurism in children and in youths is a result of acute rheumatic fever. They are rare and happily have a marked tendency towards spontaneous recovery. It is perhaps enough to realize the existence of these non-syphilitic infections of the aorta, and in a case of aortitis, occurring after one of the above infections, the possibility of its non-syphilitic origin should be considered.

*Arteriosclerosis* is often combined with syphilis in

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<sup>13</sup> REID, W. D.: *Boston Med. and Surg. Jour.*, clxxxiii, No. 3:67, and 4:105 (July 15 and 22, 1920).

<sup>14</sup> BENNERT, R.: *Zeitsch. f. klin. Med.*, Bd. lxix, Nos. 1 and 2, 121.

elderly patients. Pure atheroma rarely causes insufficiency of the aortic valve and practically never leads to the formation of an aneurism. Pain is present in but a small minority of the arteriosclerotic cases. The gross pathological findings differ, particularly in that there are fatty and calcareous changes as contrasted to the fibrosis of syphilis. The Röntgen findings of the aorta show no bulging to the right of the ascending aorta, and less dilation of the arch, but rather a lengthening of the same with a prominence of the knob to the left.

*Hypertensive heart disease* also causes enlargement of the heart and some dilation of the aorta, but may be dismissed with the comment that neither high blood pressure nor evidence of impaired renal function form part of the picture of cardiovascular syphilis.

*Rheumatic heart disease* at times must be considered. This is especially true if, in addition to the findings of aortic insufficiency, there is an apical murmur which may be explained, either as that described by Austin Flint, or as that of true stenosis of the mitral orifice. Organic change of the mitral valve is probably never syphilitic, and, if present, the insufficiency of the aortic valve may then also be of rheumatic origin. Evidence of enlargement of the left auricle and the electrocardiographic findings of mitral stenosis (which see) indicate the latter condition. True mitral stenosis, furthermore, tends to distinctly lessen the Corrigan quality of the pulse. The history is often of assistance; "apparently well until recently" and the absence of the story of an infection which might cause rheumatic heart disease, is not the history of a rheumatic heart. Nevertheless there are cases in which a distinct doubt must

remain. Resort to the therapeutic test of antisyphilitic treatment is then justified.

Heart-block may likewise raise the question as to whether it be of rheumatic or luetic causation. The therapeutic test is indicated in these cases also.

Cases in a sanatorium for *pulmonary tuberculosis* have not infrequently first been correctly diagnosed cardiovascular syphilis, as a result of radiographic examination.

*Mediastinal tumors* may at times offer some difficulty in differentiation. A careful study, with a consideration of the data present, and especially the X-ray findings, will almost always enable the correct diagnosis to be made.

*Tabes dorsalis* hardly needs differentiation save as regards the so-called cardiac crises in tabetic patients. As syphilitic infection of the heart and aorta is very common in the victims of *tabes dorsalis*, the practical importance of separating the cardiac crises in *tabes* from the anginal attacks of cardiovascular syphilis, if they be not one and the same condition, is much lessened.

**PROGNOSIS.**—On the whole the prognosis of syphilitic infection of the circulatory tissues is grave, because of the tendency to progressive impairment of the integrity of the heart and aorta. *Angina pectoris*, with its danger of sudden death, may ensue. About 30 per cent. of the recognized cases (figures are of little value, as cardiovascular syphilis often escapes recognition) are estimated to develop aneurism or insufficiency of the aortic valve or both. A fatal hemorrhage from perforation of the aneurism is not unusual.

On the other hand, evidence is now being accumulated that modern antisyphilitic therapy is being attended with

promising results.<sup>15</sup> The improvement is in symptoms and not in physical signs. Thus, diminution of the dilation of the arch of the aorta does not occur. Recollection of the pathology of cardiovascular syphilis makes it easy to comprehend why aneurism, aortic insufficiency, etc., cannot be removed. The average length of life in cases of a comparatively advanced type, from the time treatment was instituted,<sup>16</sup> was one year where mercury and potassium iodide were employed, and three years when diarsenol, or its equivalent, was added.

Considerable depends upon the amount of damage already incurred before the diagnosis is established and treatment instituted. Thus, if insufficiency of the aortic valve has ensued, serious mischief has been achieved. Cardiac failure, especially if advanced to the stage of edema of the lower extremities, always offers a poor prognosis.

**TREATMENT.**—This is primarily that of syphilis. Since it has been demonstrated that the spirochetes actually invade the heart and the aorta, the treatment should be directed toward destroying the invading organisms as quickly as possible. For this purpose arsphenamine, or its equivalent, and mercury are most useful. Syphlographers, however, differ in the details of the administration of these drugs. The relative merits of the methods advised by different authorities need not be entered into for the purposes of this book. A single method will be described which is suitable for the treatment of cardiovascular syphilis and which, in addition, appears to be attended with a minimum of risk and has been observed to obtain favorable results.

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<sup>15</sup> REID, W. D.: *Jour. Amer. Med. Assn.*, lxxiii: 1832, 1919.

<sup>16</sup> Ibid.

*Arsphenamine*, or a similar arsenical, should be injected intravenously in an initial dosage of about 0.15 gm., and, if well tolerated, rapidly increased to 0.5 gm. per dose. This latter amount should rarely be exceeded, as with this precaution, cardiac cases may be treated with but small risk of the occurrence of an unpleasant reaction. The course of arsphenamine should include six to ten injections at weekly intervals. At the same time the patient should receive mercury by mouth; suitable preparations are the *protiodide of mercury* gm. 0.01 to 0.015 (gr.  $\frac{1}{6}$  to  $\frac{1}{4}$ ), and mercury with chalk gm. 0.06 to 0.12 (gr. 1 to 2) *t.i.d.*, p.c. On completion of the course of arsphenamine and mercury, treatment is continued by a series of fifteen intra-muscular injections of mercury given at one week intervals. For this the *salicylate of mercury* in a dosage of gm. 0.065 to 0.13 (gr. 1 to 2) is a good selection.

After the twenty to twenty-five weeks of treatment, outlined above, have been given, the patient is allowed an intermission of four to eight weeks, and then the course of arsphenamine and mercury is repeated. It is sometimes permissible to replace the mercurial injections by the oral administration or by inunctions of the drug.

There may be times when, for certain reasons, it is not deemed possible to treat the patient by the above-mentioned courses of injections. In such cases mercury can at least be administered orally. Satisfactory preparations have been mentioned. As a rule it would seem that to-day mercury is rarely prescribed without the precaution of special care of the teeth, but in addition it should be emphasized that attention be directed towards the diet. Mercurial preparations often produce diarrhea of such severity

that the patient refuses to continue the use of the drug. This can usually be avoided by prescribing a diet which excludes the articles of food known to stimulate bowel action, *i.e.*, fruits, green vegetables, coarse cereals, etc. The latter may be reintroduced if the drug is being well tolerated.

*Iodide of potassium* is probably of use only in the cases coming under treatment years after the initial infection. It may be prescribed in doses of gm. 0.6 to 1.3 (gr. 10 to 20) *t.i.d.*, *p.c.*, for alternate two weeks over long periods. Some syphilographers prefer that the amount of the iodide should not exceed the smaller of the doses listed above.

No patient should be dismissed as cured, but advice should be given that he report at six to twelve month intervals for re-examination and an opinion as to the need of further treatment. A return of active symptoms or evidence of a progression of the syphilitic lesions, regardless of the Wassermann reaction, should be an indication for the resumption of full treatment.

Treatment may be further discussed according to the type of case.

1. Cases in which the *cardiac symptoms* are present within a few weeks or months of the primary lesion of syphilis. The patient should be restricted to bed and antiluetic therapy instituted. There appears to be less danger of untoward results if mercury is administered first and followed within two or three weeks by a course of arsphenamine. The patient may be allowed up after treatment has been carried out for two to three weeks and there is evidence of improvement. The further treatment is that described above.

2. Cases coming under treatment years after the primary infection and exhibiting *moderate physical signs* with but little evidence of cardiac failure. The courses of arsphenamine, mercury, and potassium iodide, as already outlined, are indicated. Many feel that the maximum dose of arsphenamine should not exceed 0.3 or at the most 0.4 gm. If angina pectoris is present, the measures suitable for that condition (see later chapter) should be added. The general hygienic measures recommended (see chapter on treatment) for the cardiopath should be prescribed.

3. Cases with *aneurism*, heart-block, marked symptoms of heart failure, etc. If the heart failure is severe, treatment should first be directed towards its relief. The measures to be employed do not differ from those suitable for the treatment of heart failure of non-syphilitic origin. After some measure of control of the cardiac embarrassment has been obtained, treatment against the syphilitic infection should be instituted. It will usually be safer to commence with mercury. After two to three weeks it may be proper to administer arsphenamine, starting as low as gm. 0.05 to 0.1 and not exceeding perhaps gm. 0.3 Mercury, in the form of pills or as an inunction, and iodide of potassium have the same indications as described above.

Discussion of the special treatment of aneurisms will be postponed to a subsequent chapter.

## CHAPTER X

### ARTERIOSCLEROTIC HEART DISEASE

ARTERIOSCLEROTIC heart disease is sometimes termed the senile heart. When considered in this sense, it can hardly be termed a disease, but may better be considered a process of involution. Since symptoms may occur and since a fair proportion of the cases occur a little before the onset of old age, it seems better for clinical purposes that arteriosclerotic changes should continue to be regarded as one type of heart disease.

It has long been the custom to include cases with high blood pressure, namely, cases with some form of hypertensive heart disease, under the term of arteriosclerosis, but, as so strongly urged by Allbutt, it no longer seems intelligent to do so.

**ETIOLOGY.**—Apparently there are so many factors involved in the production of arteriosclerosis that it is hardly possible to speak with assurance of any one. When the condition is not evident until old age, it is customary to state that the arteriosclerotic changes are due to the advanced years and the wear and tear of life.

In recent years, evidence, some of which is confirmed by experimental work, has accumulated that pneumonia, typhoid fever, the exanthemata, and, in fact, most of the infectious diseases, are followed by some degree of arteriosclerotic changes. Excessive mental strain<sup>1</sup> would appear to be a potent factor. The absorption of lead is held by some to produce sclerotic changes.

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<sup>1</sup> MÜLLER, FRIEDRICH: *Arch. Int. Med.*, 1 : 1, 1908.

**PATHOLOGY.**—In the early stages the intima shows yellowish placques or streaks which take the stain for fat. Then bluish-white, translucent connective tissue forms round the placque. MacCallum<sup>2</sup> describes these as having the appearance of drops of paraffin on the intima. The internal elastic lamella under the placques is often frayed and the smooth musculo-elastic layer of the intima may be destroyed. The centre of the placque is soft, and, if the roof breaks through, the contents are washed away in the blood stream, with the resulting production of an atheromatous ulcer. Thrombus formation is then likely. Deposits of cholesterol and the phosphates of calcium and magnesium are found in the process.

In the main the media is not much involved save in the smaller arteries, where some thinning and fat deposit are found. The type of sclerosis described by Monckeberg,<sup>2</sup> which may be a different process from true arteriosclerosis, occurs mostly in the arteries of the leg. In this form of sclerosis masses containing fat, lime, and even bone with marrow, are found in the media. The intima does not always show thickenings over it, though the type of intimal sclerosis described above may occur along with Monckeberg's sclerosis.

The aorta is usually dilated and lengthened so that it curves from one side to the other of the vertebral column. In the branches, the placques may distinctly diminish the calibre of the vessel by encroaching upon the lumen. At times large masses are present which practically obliterate the lumen of the arteries and produce gangrene.

<sup>2</sup> MACCALLUM, W. G.: Text-book of Pathology, W. B. Saunders Co., Philadelphia, 1916, p. 322.

<sup>2</sup> *Ibid.*

MacCallum states that these may or may not be organized thrombi.

The heart tends to be small, and the myocardium thinner than usual. The heart muscle is often of a brownish hue, the so-called "brown atrophy," and feels flabby to the fingers. On histological examination the brown atrophy may be recognized by the brown pigment granules. Fragmentation of the muscle fibres is common. In some cases, in which hypertension is associated with arteriosclerotic heart disease, the pathology shows a mixed picture; *i.e.*, there is some hypertrophy of the muscle fibres and areas of replacement by fibrosis.

The heart valves, especially the aortic and mitral, may become thickened and puckered. Deposits of lime in these valve thickenings are common.

**SYMPTOMS AND SIGNS.**—Varying degrees of a lowering of the power of the heart may be observed. In the average case, the diminution of the cardiac strength comes on gradually and is only a part of the general decline of the physical powers that occurs in normal old age. Elderly persons show a distinct tendency to lessen their physical activities, and it may be only as a result of some sudden or unusual exertion that attention is drawn to the heart. Occasionally the picture of angina pectoris may be present.

Arteriosclerotic heart disease is commonly but part of general arteriosclerosis; certain symptoms, not primarily cardiac in origin, are therefore often present in these patients. A change in the mental faculties, varying from a lessening of the power of memory to the condition of senile dementia, may be observed. A tendency to headache is frequent. The latter has been explained on a circulatory

basis, as due to a congestion in the cerebral circulation.<sup>3</sup> Dizziness, on change from the horizontal to the upright position, or on stooping, is due to a sluggishness of the vasomotor reflex, by which, in the normal individual, a physiological adjustment is promptly made for the maintenance of a normal circulation in varying postures of the body. Discomfort or pain in the calf of the leg on walking appears to be due to the failure of the increase in blood supply needed in the leg muscles during exercise. Other symptoms of general arteriosclerosis need not be mentioned here.

On physical examination the peripheral arteries show changes to palpation. The wall feels thickened and more rigid, and in cases where deposition of lime salts has occurred, the artery may give a beaded sensation to the palpating finger. A marked degree of the latter condition is sometimes termed the "pipe-stem artery." The blood pressure is usually not essentially changed, ranging from a little below to a little above the normal adult level. It is a mistake to hold that any degree of increased blood pressure is the rule in arteriosclerosis, as in by far the majority of such patients the blood pressure readings are within normal limits.

An increase of the supraventricular dulness, as well as a visible or palpable pulsation in the suprasternal notch, may often be noted. They are produced, of course, by the elongation and consequent change in the position of the aorta, which occurs in arteriosclerosis.

The Röntgen examination typically discloses a heart

<sup>3</sup> INMAN, T. G.: "Cerebral Thrombosis and Abrupt Slowing of the Cerebral Circulation," *Jour. Amer. Med. Assn.*, 75, 26: 1765 (Dec. 25, 1920).

slightly smaller than normal and a prominence of the aortic knob to the left.

By the usual clinical methods of examination, the heart appears to be of normal size or sometimes a little small. The frequent concomitance of emphysema of the lungs may obscure the borders of the cardiac dulness. The sounds are likely to be somewhat less loud than in younger individuals. The second aortic sound is commonly of a duller quality, perhaps due to a thickening of the semilunar cusps and adjacent tissues. A systolic murmur, generally soft in quality, often accompanies the apical first sound, and a louder, rougher murmur, transmitted outward and to the neck, may be associated with the first sound at the second right costal cartilage. The diastolic murmur of an insufficiency of the aortic valve is relatively rare.

Extrasystoles, or premature beats, are common in the arteriosclerotic heart. Less commonly, fibrillation of the auricles will be found to be the cause of an irregularity of the cardiac rhythm. Heart-block is a still further complication. The latter two are often present at the same time.

**DIAGNOSIS.**—The appearance of symptoms of an insufficient circulation in an elderly patient, or in one in whom the general or peripheral signs of arteriosclerosis are present, should immediately draw attention to the heart. The dull quality of the second sound at the aortic area is helpful but not diagnostic, as a similar change may be found in cardiovascular syphilis. The murmurs should not mislead one, as they are merely part of the picture of the arteriosclerotic heart, and do not indicate an infection such as that occurring in rheumatic heart disease. It should be remem-

bered that certain of the arrhythmias, *i.e.*, premature beats, auricular fibrillation, and heart-block, are common in arteriosclerotic disease. Angina pectoris, appearing in a patient over fifty-five years of age, points to arteriosclerotic changes in the heart, as a rule. If the blood pressure is persistently elevated, the condition is probably not true arteriosclerotic heart disease, but some form of hypertensive heart disease.

**PROGNOSIS.**—It is not to be expected that arteriosclerotic changes in the circulation should disappear. A slow progression is the rule. Those cases which make their appearance before true old age, as, for example, those which appear to be the result of infections or severe nervous strain, tend to advance to a considerable degree of circulatory impairment. The general deterioration of the patient can be considerably checked by judicious treatment.

**TREATMENT.**—The first essential is an intelligent regulation of the patient's life. All chafing at the lessening of the physical and mental powers is to be discouraged and combated by tactful explanation of the futility of the same, and conversation directed towards a philosophical acceptance of the situation. Experience has shown that elderly people are benefited by association with young people. It tends to keep them more cheerful and interested in life.

Daily exercise of a light character will do much to maintain the general health. Walking and light golf are examples of suitable exertion.

An extra amount of rest is indicated. There is reason to believe that such patients do better and have less head-

ache from cerebral congestion<sup>4</sup> if the sleeping periods are not too long, and therefore the patient should not be encouraged to remain in bed late in the morning. One or more periods during the day may be arranged for a nap.

*Diet.*—Since the teeth are usually absent or deficient, the food should be such that does not require much chewing. In consideration of the fact that there is less hydrochloric acid in the gastric juice, and digestion is slower, the food should be simpler, moderate in amount, and the meals preferably not less than four hours apart. In certain respects the diet should revert towards that suitable in early life.

Drug treatment is of secondary importance. At times drugs may be used to relieve symptoms. Strychnine, quinine, arsenic, etc., are often prescribed as tonics. Various bitters, as stomachics, and laxative medicines are often used. In deciding whether to give a medicine, it seems wise not to overlook the favorable psychic effect when dealing with a patient who believes strongly in drugs.

*Digitalis.*—It is doubtful if this drug is of any benefit to the arteriosclerotic heart, if normal rhythm is present. If given, it should be prescribed tentatively and in small dosage. Decision as to the continuance of the drug may be based on the effect observed.

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<sup>4</sup> INMAN, T. G.: "Cerebral Thrombosis and Abrupt Slowing of the Cerebral Circulation," *Jour. Amer. Med. Assn.*, 75, 26: 1765 (Dec. 25, 1920).

## CHAPTER XI

### HYPERTENSIVE HEART DISEASE

THIS title applies to a group of conditions in which the essential feature is a persistent elevation of the blood pressure. There are three types which are often sufficiently distinct to permit recognition. The first, for which the term "chronic vascular hypertension" seems satisfactory, has been described in the literature under other names, *i.e.*, Allbutt's hyperpiesis, essential or primary hypertension, chronic hypertensive cardiovascular disease, arteriosclerosis, etc. The second type, "chronic hypertension with diabetes," usually of mild degree, differs little save in the disturbance of the sugar metabolism. The third form is satisfactorily labelled, it would appear, "chronic hypertension with nephritis." Although these three types may usually be distinguished by careful study, and it has been urged that at least the first and third form obtain separate classification, it would appear consistent with the cardiac aspects, and more in accordance with the present state of our knowledge, to resist the temptation to break up the group.

**ETIOLOGY.**—The age of the patient varies from as low as twenty-five years to old age, with a marked predominance of the decades forty to fifty and fifty to sixty. The causation of hypertensive heart disease is as yet obscure. Overeating and mental strain or worry appear to be provocative agents. Disturbances of the ductless glands, particularly the condition of hyperthyroidism, is often associated. The importance of a change in the function

of the ovaries has been emphasized by Hopkins,<sup>1</sup> who believes that there is a distinct type of hypertension occurring at the menopause. That this latter should be separated from the larger group is uncertain. The rôle of certain poisons and infections is not yet clear. The development of the studies in the chemistry of the blood has brought to light that the salt metabolism is nearly always disturbed. The threshold for the excretion of salt is high.<sup>2</sup> Judgment as to the etiological importance of certain factors is often hampered by the fact that many cases are not noted until the patient has reached the age at which appear the arteriosclerotic changes associated with the onset of old age. In fact, it is only of recent years that an attempt has been made to separate chronic vascular hypertension from involutionary arteriosclerosis.

**PATHOLOGY.**—In the early or presclerotic cases, autopsy may fail to disclose arterial or renal disease. The hypertension appears to be functional, due to a hypertonus of the arterial musculature, thereby producing a narrowing of the lumina of the arterioles in the precapillary area. In the majority of cases, within a few months, to one or two years, there ensue sclerotic changes in the arterial walls and hypertrophy of the myocardium. The arterial changes do not differ essentially from those found in arteriosclerosis without hypertension. The kidneys commonly show moderate change of the character occurring in arteriosclerosis. The renal changes may be somewhat more marked in the cases diagnosed clinically hypertension with nephritis.

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<sup>1</sup> HOPKINS, A. H.: "Climacteric Hypertension," *Amer. Jour. Med. Sci.*, 6, clvii: 826, 1919.

<sup>2</sup> ALLEN, F. M.: "Arterial Hypertension," *Jour. Amer. Med. Assn.*, 74, 10: 652 (March 6, 1920).

The cardiac change, however, as so well emphasized by Allbutt,<sup>3</sup> is radically different from that obtaining in arteriosclerosis. The myocardium is hypertrophied rather than degenerated. It is thicker and on microscopic examination its fibres are increased in number and size. At a later stage, when cardiac insufficiency appears, the heart muscle shows necrosis of some of the muscle fibres, with replacement by fibrous tissue, a form of fibrous myocarditis. The aortic and mitral valves are not infrequently thickened and puckered. In cases coming to section, after death caused by cerebral hemorrhage, the presence of the latter will, of course, be added to the post-mortem findings.

**SYMPTOMS.**—Many of these cases are first found in the course of a routine physical examination which has been prompted because of some intercurrent disease. The individual often appears robust but in a state of overtension. The type has been pictured thus: "The patients are overweight and sometimes even obese. The neck is short, the muscles are soft, their bodily movements are sluggish, their carriage and walk are ungraceful, and they lack the spring and élan of the former athlete. Physically these people are tense; they pursue their vocation with tremendous seriousness and worry over trivialities. Phlegm and hypertension are, in my experience, antagonistic. Furthermore, these patients have narrow intellectual horizons. Their interest in anything outside of their business is desultory. They have no hobbies."<sup>4</sup>

Gastric symptoms may lead the patient to consult a

<sup>3</sup> ALLBUTT, C. A.: Diseases of the Arteries, Including Angina Pectoris, 1915, Macmillan & Co., Ltd.

<sup>4</sup> MOSCHOWITZ, E.: "Hypertension: Its Significance, Relation to Arteriosclerosis and Nephritis, and Etiology," *Amer. Jour. Med. Sci.*, 158: 668 (Nov., 1919).

physician. These complaints are of the type consistent with gastric neurosis; gas formation and constipation may be prominent. In other individuals nervous symptoms and pains in the legs are conspicuous. Headache is common. A little shortness of breath and evidence of cardiac failure may make their appearance. In more advanced or complicated cases, the signs and symptoms of marked heart failure, angina pectoris, or cerebral hemorrhage may be present. Weakness, vertigo, and epistaxis are still further symptoms. Patients in whom hypertensive heart disease is associated with nephritis appear sicker and often have an anemia in addition. Edema is a more frequent condition in this type of hypertensive heart disease.

**PHYSICAL SIGNS.**—The sphygmomanometer shows a constant elevation of the systolic and diastolic blood pressures; the former may be as high as 300 or more, while the latter may even reach 190. While it is known that the blood pressure varies in accordance with age, sex, and other factors, it seems a reasonable rule to consider hypertension present if the systolic pressure exceeds 150 or 160 in men, and 140 or 150 in women, and the diastolic above 100 in either.

Examination of the heart may give normal findings, but if hypertrophy has ensued the apex impulse is usually displaced downward and to the left, and, other things being equal (*i.e.*, the contact of the heart with the chest wall, the thickness of the parietes, etc.), is more forcible in character. The area of deep cardiac dulness is increased in accordance with the extent of the left ventricular enlargement. The width of the supraventricular dulness commonly shows an increase due to the broadening of the

arch of the aorta. On auscultation the first sound at the apex is usually of a more booming character and the second sound at the level of the second right costal cartilage is accentuated. There are no murmurs characteristic of hypertensive heart disease. A systolic murmur at the apex may indicate an insufficiency of the mitral valve, of muscular causation, and a systolic murmur at the aortic area may result from the relative dilatation of the first part of the aorta. The rhythm is normal, but it may be interrupted by an abnormal rhythm, especially by premature beats, as in other types of heart disease.

Ophthalmoscopic examination may show sclerosis of the retinal arteries and occasionally hemorrhage in addition.

Urinary examination generally discloses the presence of albumin varying in amount from a very slight to a large trace, with a few to many hyalin and granular casts. The tests of the functional power of the kidneys demonstrate a difference between the types of hypertensive heart disease. In the first two, *i.e.*, chronic vascular hypertension and hypertension with diabetes, there is commonly no reduction in renal function save in the handling of sodium chloride.<sup>5</sup> The chloride content of the blood plasma and the renal threshold of salt excretion are both high. In the cases of hypertension with nephritis, these tests will disclose, in addition, some fixation of the specific gravity of the urine, a diminution in the ability to excrete phenolsulphonephthalein, and an increase in the blood urea content, etc. When diabetes is associated with hypertensive heart disease, sugar may be detected in the urine, or the blood chemistry may show abnormalities in relation to sugar.

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<sup>5</sup> ALLEN, F. M.: "Arterial Hypertension," *Jour. Amer. Med. Assn.*, 74, 10: 652 (March 6, 1920).

Röntgen examination may give normal cardiac outlines, or, if hypertrophy has occurred, the heart shadow is increased in its long diameter and in the region of the left ventricle. The width of the great vessels shows moderate increase.

The electrocardiogram in a typical case shows left preponderance, often of marked degree.

**DIAGNOSIS.**—The earliest and essential point is the elevation of the blood pressure. The decision that true hypertension is present should be based on the finding of high pressures on examination with the sphygmomanometer at several different occasions, and after due allowance has been made for temporary elevation caused by transient emotional factors. Judgment should likewise be withheld as to the presence of hypertensive heart disease if the patient is suffering from some febrile disease, such as pneumonia, which may depress the blood pressure below its normal level. Evidence of hypertrophy of the left ventricle is of distinct confirmatory value.

A test for the rate of the metabolism is frequently of value. If the patient is at the time of the menopause, a disturbance of the balance of the ductless glands is to be considered.

Studies of the renal function and of the blood chemistry are necessary in order to differentiate the individual types of hypertensive disease of the heart. The fact that cases occur in which the findings are intermediate between the three forms of hypertension serves to emphasize that an absolute separation would be purely arbitrary.

In the late stages, with marked evidence of cardiac failure, the effect of treatment will do much to differentiate

whether the heart or the kidney is the primary cause. If the heart responds well to digitalization, the case is cardiac rather than nephritic. If edema is present, three doses of theobromin sodium salicylate (diuretin) 1 gm. (gr. xv), or theophyllin sodioacetate (theocin) 0.3 gm. (gr. v) every four hours for four to six doses, may be given. If the kidney responds well the underlying condition is not nephritic.

Arteriosclerosis is readily excluded by its essentially normal blood pressure. Cardiac hypertrophy is not a feature of arteriosclerosis. The latter may, however, be present in addition to the hypertensive heart disease.

**PROGNOSIS.**—Since the nature of hypertension is not yet clearly understood it follows that the prognosis in individual cases cannot be depicted with certainty. Experience, however, has demonstrated that the prognosis varies according to the type of hypertensive heart disease.

In chronic vascular hypertension, judicious treatment, if undertaken early, may arrest or materially check the process so that the patient may live in comparative comfort for many years. If the patient is much overweight, accustomed to overindulgence in food, or subject to excessive nervous strain, there is greater prospect of benefit from treatment than there is in patients exhibiting less evident points of attack. When the condition does not yield to treatment there is always danger of sudden death from cerebral hemorrhage or less frequently from angina pectoris. A more gradual death from cardiac insufficiency terminates probably the majority of cases, and in the experience of the writer, this form of heart complication is more frequent than is angina pectoris. A few cases

progress to marked impairment of the kidneys and succumb to uremia.

Hypertensive heart disease with diabetes often does well on the dietetic treatment of diabetes or obesity. In the main, its prognosis appears similar to that of chronic vascular hypertension.

The third type, namely, hypertensive heart disease with nephritis, has the least favorable prognosis. A return to normal health is unusual; some degree of anemia and weakness usually persist. Within a few years the majority of such patients succumb to progressive impairment of the kidney, and uremia.

TREATMENT must be based upon an accurate diagnosis, with a recognition if possible of the type of hypertensive disease. Granting that it may not be possible to state just what caused the heightened blood pressure in an individual case, it seems reasonable to lessen those factors which the study of physiology has shown cause a rise in the blood pressure level.

The early and less advanced cases should be treated by modification of their life so as to reduce the *intake of food* and the outgo of nervous energy. In the diabetic type the diet suitable for a case of mild diabetes is usually beneficial. If evidence of kidney impairment predominates, the dietetic measures suitable for nephritis should be followed. In all cases the amount of food should be low.

The *salt intake* is of special importance. In many cases it is enough to reduce the amount of salt, but if the hypertension persists it may be necessary to carefully arrange a diet in which the salt does not exceed 2 gm.

(30 gr.) per day.<sup>6</sup> In the nephritic type, however, it may sometimes be necessary to give more than 2 gm. (30 gr.) for its diuretic effect and the prevention of uremia. In the cases, then, which do not yield to simpler measures, it is doubly indicated to obtain the help of chemical analysis of the blood and to guide the treatment largely by its findings. Further discussion of this is beyond the scope of this book; it is more the treatment of nephritis than that of cardiac disease.

The amount of *fluid intake* should usually be reduced. It is true that some patients do well on two to three litres per day, but many are benefited by keeping the amount down to one or one and a half litres, or less. A recent report,<sup>7</sup> however, announces very favorable results from the addition of water to the daily intake.

Constipation, if present, should be relieved, preferably by the non-drug methods, *i.e.*, regular time for going to stool, diet, suds enemata, liquid petrolatum U.S.P., etc. Although it is not good practice from the standpoint of the treatment of the constipation problem, there is much clinical evidence that patients suffering from chronic vascular hypertension are benefited by an occasional dose of salts, castor oil, or calomel.

The condition of tension and mental strain should be met by ascertaining its cause and removing the latter if possible. Herein lies an opportunity for the physician to use all his tact and diplomacy in correcting by judicious conversation the patient's philosophy of life, which is nearly

<sup>6</sup> ALLEN, F. M.: "Arterial Hypertension," *Jour. Amer. Med. Assn.*, 74, 10: 652 (March 6, 1920).

<sup>7</sup> ORR, J. B., and INNES, I.: "The Effect on Arterial Hypertension of Increased Fluid Intake," *Brit. J. Exper. Path.*, 3: 61, 1922.

always faulty. The patient exhibiting hypertensive heart disease will be found in the majority of cases to be at odds with his environment. It is often helpful if the patient can be induced to read one or more of the little books written by Dr. George Lincoln Walton.<sup>8</sup> Much good may result if the play element can be introduced into the patient's life. A vacation is often indicated. The amount of work done by the patient may well be reduced by persuading him or her to turn over many of the minor items to subordinates. Some form of light outdoor exercise is generally beneficial. Golf and walking are examples.

A warm cleansing bath every other night, with a warm salt bath, of seven to ten minutes' duration, on the alternate nights, produces a general vascular relaxation and promotes sleep. Temporary use of the milder sedatives and hypnotics may be resorted to in cases manifesting excessive nervous tension.

If the rate of metabolism is high, treatment suitable for hyperthyroidism should be added. In cases occurring in women at the time of the menopause, a trial may be made of corpus luteum, or extract of the whole ovarian gland.

In the more severe or advanced cases, rest in bed for two to three weeks, or, less safely, sweat baths may be tried. Symptoms are to be met. Venesection, with the removal of 350 to 500 or more c.c. of blood, appears to benefit intractable cases. Such a procedure repeated at intervals of three to twelve months has been followed by considerable relief in selected cases. Nevertheless, it should not be

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<sup>8</sup> "Why Worry," J. B. Lippincott Company, 1908.

"Those Nerves," J. B. Lippincott Company, 1909.

"Calm Yourself," Houghton, Mifflin & Co., 1913.

resorted to indiscriminately, and should in no wise cause a relaxation in the therapeutic measures already mentioned.

While drug treatment is less satisfactory for cases of hypertensive heart disease, it seems at times to be indicated, particularly for the relief of symptoms. The nitrites, especially nitroglycerine, are employed, and more recently benzyl benzoate. (For further details on these drugs and the therapy of angina pectoris, the reader is referred to the chapter on the latter condition.)

Severe cardiac failure, edema, or the various arrhythmias are to be treated essentially the same as when present in heart disease of other causation.

## CHAPTER XII

### THE HEART IN HYPERTHYROIDISM

THE heart in hyperthyroidism exhibits signs and causes symptoms sufficiently distinctive to warrant its description as a cardiac entity. It may be said that symptoms referable to the heart are present at some time in practically every case of hyperthyroidism.

**ETIOLOGY.**—It is generally held that the condition is due to the action of the secretions of the abnormal thyroid gland directly on the myocardium or on the nerves controlling the heart. A suspicion exists, however, that secondary infections may play an important part. It has been noted in the laboratory, that animals suffering from hyperthyroidism, produced experimentally, are especially prone to incur various infections. This fact naturally suggests the possibility that many of the cardiac changes in man may be the result of infections.

**PATHOLOGY.**—This is not well understood, perhaps because too few hearts of patients suffering from hyperthyroidism have been subjected to careful histological study. Goodpasture<sup>1</sup> reports two cases in which myocardial necrosis was demonstrated. The process was very extensive and diffuse in one, and limited and focal in the other. He ascribes the necrosis of the muscle cells as due to a toxic myocarditis and not to occluding vascular lesions or local infection. A similar condition, though rare in adults, occurs in children in association with extreme intoxication, usually by diphtheria or scarlet fever. Good-

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<sup>1</sup> GOODPASTURE, E. W.: *Jour. Amer. Med. Assn.*, 76, 23:1545, 1921.

pasture, however, believes it is better perhaps to regard the necrosis as a result of an injurious agency other than that responsible for the underlying cardiac disease.

The gross post-mortem appearances of the heart in hyperthyroidism are dilatation of the right chambers, moderate hypertrophy of the left ventricle, and slight fatty change. Goodpasture,<sup>1</sup> again, is authority for the statement that there are few instances on record in which actual destructive lesions of heart muscle have been observed in exophthalmic goitre, even in those dying of cardiac failure.

**SYMPTOMS AND PHYSICAL SIGNS.**—In early cases, a tachycardia is present, but is unaccompanied by signs of myocardial change. With persisting rapid heart action, enlargement may become evident. An increased pulsation over the right ventricle is usually to be observed. Progressive enlargement and overaction is followed by damage to the myocardium, which may be manifested by the appearance of an arrhythmia, such as auricular fibrillation, auricular flutter, heart-block, etc. Sinus arrhythmia and premature beats may also be in evidence. Hypertension occurs in some cases of hyperthyroidism.

The tachycardia of hyperthyroidism persists day and night. It is usually between 90 and 120, and is subject to considerable variation in rate following exercise, excitement, rest, etc.

It is not to be supposed that the heart in hyperthyroidism can be satisfactorily understood unless the observer is familiar with hyperthyroidism as it affects the patient as a whole. In the absence of laboratory support the cardiac symptoms should rarely be ascribed to hyperthyroidism

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<sup>1</sup> GOODPASTURE, E. W.: *Jour. Amer. Med. Assn.*, 76, 23: 1545, 1921.

save in the presence of several of the main symptoms, such as: tremor, thyroid enlargement, exophthalmos, retraction of the upper eyelid, loss of weight, increased appetite, pronounced motor restlessness, emotional instability, or sensations of heat, undue sweating, diarrhea, polyuria, etc.

Hypersensitiveness to epinephrin may be noted in many patients, but not in all with hyperthyroidism, and it also occurs in others in whom this disease may be excluded. The clinical significance of the reaction is not clear, but at present it should certainly not be regarded as having any specific significance in the diagnosis of hyperthyroidism.<sup>2</sup>

The determination of the basal metabolism, where facilities therefor are available, offers very valuable assistance in the diagnosis of hyperthyroidism. Save for a moderate elevation in fever of the rate of the basal metabolism, the finding of an increased rate indicates hyperthyroidism. In the main there may be said to be two types of hyperthyroidism; in one, the sympathetic type, the pulse rate is elevated to a greater degree than the rate of the metabolism, and in the other, the vagotonic type, the reverse holds true. The prognosis is better, according to Means,<sup>3</sup> in the sympathetic type.

The electrocardiographic findings have been discussed by Krumbhaar,<sup>4</sup> and White and Aub.<sup>5</sup> There are no data of pathognomonic significance. The T wave is usually of increased amplitude, but apparently there is a very limited parallelism between the height of this wave and the

<sup>2</sup> PEABODY, F. W., STURGIS, C. C., THOMPKINS, E. M., and WEARN, J. T.: *Amer. Jour. Med. Sci.*, 161, 4: 508 (April, 1921).

<sup>3</sup> MEANS, JAMES H.: *Med. Clin. No. Amer.*, 3, 4: 1092 (Jan., 1920).

<sup>4</sup> KRUMBHAAR, E. B.: *Amer. Jour. Med. Sci.*, 1918, 155, 175.

<sup>5</sup> WHITE, P. D., and AUB, J. C.: *Arch. Int. Med.*, 1918, 22, 766.

basal metabolism. The electrocardiogram has, of course, its usual value in the accurate diagnosis of and the permanently recording of the various arrhythmias that may affect the heart.

In patients in whom the hyperthyroidism has ceased spontaneously or following treatment, the heart may show evidence of permanent damage. Thus, auricular fibrillation or some other functional disturbance may be present. In such cases, the signs and symptoms of cardiac impairment may not differ from those present in disturbances of other causation, and their connection with a previous hyperthyroidism may be appreciated only by the history of the occurrence of the latter condition.

**DIAGNOSIS.**—According to Friedrich Müller,<sup>6</sup> about one third of the cases affected by moderate palpitation are of thyroid origin. The diagnosis depends upon some of the above-mentioned signs and symptoms of abnormality of the heart, in association with evidence of hyperthyroidism. An unstable tachycardia is strongly suggestive of hyperthyroidism. Electrocardiographic evidence may be helpful. An elevation of the rate of the basal metabolism proves the diagnosis.

Paroxysmal tachycardia differs in that it is usually of limited duration, of sudden onset and ending, and the heart rate in an individual attack varies not more than a few beats for change of posture, exercise, etc. Vagal pressure and the manœuvres which abolish some attacks of paroxysmal tachycardia will not so affect the tachycardia of hyperthyroidism.

The tachycardia of effort syndrome, cardiac neurosis,

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<sup>6</sup> MÜLLER, FRIEDRICH: *Arch. Int. Med.*, 1:1 (March, 1908).

etc., does not last day and night and gives more evidence of being of reflex origin.

After the hyperthyroidism has ceased, the history of that disease may be necessary to explain the cause of cardiac impairment.

**PROGNOSIS.**—If the existing intoxication is the chief factor in the production of the arrhythmia, the latter may disappear with successful treatment of the hyperthyroidism. As previously noted, the prognosis is somewhat better in cases in which the pulse rate is elevated out of proportion to the increase in the rate of metabolism, *i.e.*, in the so-called sympathetic type. Damage of the myocardium, evident after the cessation of the hyperthyroidism, is permanent.

**TREATMENT.**—This is essentially that of the underlying condition, the hyperthyroidism. The chief measures are rest, Röntgen radiation of both thyroid and thymus glands, and surgical measures. When auricular fibrillation is present digitalization is indicated before surgical treatment is undertaken.

There is experimental evidence<sup>7</sup> that chloroform as an anesthetic in hyperthyroidism is apt to be exceptionally detrimental to the myocardium, and should be avoided.

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<sup>7</sup> GOODPASTURE, E. W.: *Jour. Exper. Med.*, Baltimore, 34, 4:407 (Oct. 1, 1921).

## CHAPTER XIII

### THE HEART IN DIPHTHERIA

IT IS well known that the heart is frequently, and sometimes seriously, affected in diphtheria. The changes are sufficiently unique to warrant description.

**Etiology.**—This is primarily, of course, that of diphtheria, namely the Klebs-Löffler bacillus. But pathology teaches that the organisms remain at the site of membrane formation, and that the internal damage is the result of absorption of the toxin secreted by the diphtheria bacilli. It might reasonably be thought that some of the heart damage is due to the invasion of secondary organisms, in a similar manner as the streptococci, staphylococci, etc., are known to cause pneumonia in some cases of diphtheria. However, bacteria have not been found in the lesions<sup>1</sup> in the heart and the process may therefore be properly considered as a toxic myocarditis. The greater the amount of membrane and the longer the delay before sufficient antitoxin is given, the more likelihood there is of serious involvement of the heart.

**Pathology.**—This is of special interest because of the difference in opinion expressed in the literature as to whether any permanent damage remains in the hearts which have been seriously but not fatally involved. To quote one of the most exhaustive studies<sup>1</sup> of the condition, "Degeneration of the myocardium is one of the common

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<sup>1</sup> COUNCILMAN, W. T., MALLORY, F. B., PEARCE, R. M.: "A Study of the Bacteriology and Pathology of Two Hundred and Twenty Fatal Cases of Diphtheria," *Jour. Boston Soc. Med. Sci.*, v: 189-319, 1900.

conditions found in diphtheria. The simplest form of this is fatty degeneration, which is found in the majority of all cases. This varies in extent, at times affecting the myocardium generally, at times occurring in foci. . . . . The fatty degeneration accompanies and seems to precede the more advanced forms of degeneration which lead to the complete destruction of the muscle. . . . . Fragmentation and fracture of the degenerated muscle cells is often found . . . . . Simple fatty degeneration is found in the severe cases of short duration, the more extensive degenerations in the more prolonged cases. . . . .

"Acute interstitial lesions of two sorts are found. In one there are focal collections of plasma and lymphoid cells in the tissue, which may be accompanied by degeneration of the myocardium, but are not dependent upon it. This condition is analogous to acute interstitial nephritis. In the other condition, the interstitial lesion consists of a proliferation of the cells of the tissue and is secondary to the degeneration of the muscle. It is probable that this may lead to extensive formation of connective tissue and some of the cases of fibrous myocarditis may be due to this."

Thrombosis due to primary necrosis of the endocardium is not uncommon. The post-mortem findings in cases showing high-grade heart-block have been recorded.<sup>2</sup>

SYMPTOMS.—There is an initial acceleration of the heart rate to 130, 150, or more, and this usually subsides within forty-eight hours after the administration of anti-toxin. In about one third of the cases irregularities of the heart follow after the convalescence has been established

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<sup>2</sup> FLEMING, G. B., and KENNEDY, A. M.: *Heart*, ii, 2: 77, 1910.

MAGNUS-ALSLEBEN, E.: *Zeitschr. f. klin. Med.*, lxix: 82, 1910.

PRICE, F. W., and MACKENZIE, IVY: *Heart*, iii, 2: 233, 1912.

about one week. According to the statistics of S. C. Smith,<sup>3</sup> 85 per cent. of these arrhythmias are of simple nature, such as sinus arrhythmia, sino-auricular block, or premature beats. The remainder of the convalescent abnormalities were shown to be due to heart-block. The latter is often sudden in its onset and tends to become complete.

A marked fall in the blood pressure occurs in the more severe cases. After a critical study of diphtheria, one observer<sup>4</sup> concludes that an essential feature is a decrease in the total volume of the blood and also an uneven distribution of the latter. As a result, the peripheral vessels may be almost empty.

The patients in whom the serious heart lesions are present are usually those who were profoundly toxic when treatment was first instituted and in whom the initial tachycardia persisted for several days. They may be dull but fully conscious. The extremities are cold and the skin pale or sometimes slightly dusky. Pallor around the mouth is often present in such cases. Vomiting may occur. The pulse may be rapid and of poor quality, but when considerable heart-block is present the rate is usually sixty or below. Triple or gallop rhythm, perhaps indicating incomplete heart-block, may at times be noted. Death often results in one to two days. Embolism from the thrombi in the heart occurs occasionally.

Auricular fibrillation is apparently rare, but has been

<sup>3</sup> "Observations on the Heart in Diphtheria," *Jour. Amer. Med. Assn.*, 77, 10: 765, 1921.

<sup>4</sup> HARDING, M. E.: The Circulatory Failure of Diphtheria, 1920, Univ. of London Press, Ltd.

recorded.<sup>5</sup> It occurred following complete heart-block and was permanent. In this case, also, the appearance of heart-block was delayed more than seven days after the onset of convalescence, in fact it was on the twenty-third day of the disease. Later in the convalescence, the pulse may be fairly normal in frequency but evidences a tendency to accelerate excessively on the least exertion, such as rising to the erect posture. This usually passes off in a few weeks.

**DIAGNOSIS.**—Normally there is a history of recent diphtheria. In any such condition as that of acute toxic heart-block, described above, the probability of diphtheria as a cause should be suggested and confirmed by the history, in case the latter was not known to the examiner.

**PROGNOSIS.**—The laryngeal type of diphtheria is less liable to be complicated by severe degeneration of the heart. Experience shows that the weakness of the heart, or other cardiac abnormalities, offer positive symptoms and physical signs during a period of sufficient duration to make their presence known. Thus patients rarely die suddenly, in a strict sense of the term. A persistence in the initial tachycardia and much evidence of toxicity are ominous. The cases showing severe cardiac symptoms on or after the sixth day usually die within a few days. In the experience of E. H. Place,<sup>6</sup> the patients who survive heart-block for a week have a good chance of recovery, and deaths from this type never occur after two weeks. In by far the majority of cases, if the patient survives the acute attack, the heart, after a few months, is perfectly normal, even to

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<sup>5</sup> PARKINSON, JOHN: "Auricular Fibrillation Following Complete Heart-Block in Diphtheria," *Heart*, vi, 1:13, 1915.

<sup>6</sup> *Jour. Amer. Med. Assn.*, 77, 10:771, 1921.

electrocardiographic examination. Studies of the pathology, however, suggest the possibility of the replacement of some of the cellular elements by fibrous tissue. In Parkinson's case, heart-block appeared on the twenty-third day of the diphtheria and auricular fibrillation followed on the thirty-third day. The heart-block eventually disappeared, but the fibrillation, of which electrocardiograms were repeatedly obtained, proved permanent. This is the only case, known to the writer, in which permanent damage to the heart seems conclusively established.

**TREATMENT.**—Prevention is all-important by early and efficient use of antitoxin, intravenously if need be. It is foolish to wait for a positive culture if the clinical diagnosis of diphtheria is evident; at least one dose of antitoxin should be given. The aim is not to have the membrane shrivel and gradually come off in the course of a week or more, but it should be largely off within the first two or three days.

Digitalis and other heart stimulants are of no benefit and may do harm. The only treatment that is of much value is that of absolute rest in as near the recumbent posture as possible. The rest must be prolonged so as to give the myocardium time to recover from the toxic degeneration. Persistent vomiting may be met by allowing nothing by mouth, the injection of fluid by rectum, just enough to allay thirst, and the administration of morphia sufficient to keep the patient quiet and allay pain, if present.

## CHAPTER XIV

### CONGENITAL HEART DISEASE

CASES of congenital heart disease are uncommon and often most complicated. The majority are defects in development, but a few are regarded as due to an infection of the endocardium and myocardium during fetal life. The signs and symptoms are so variable and the possible abnormalities in the heart are so numerous and bizarre that an accurate diagnosis is frequently impossible.

SYMPTOMS may be absent. Cyanosis is often present. It varies from a cyanosis that is limited to the lips, ears, etc., to one that is general. The skin may be almost purple, and of a far more intense color than occurs, with rare exceptions, in the cyanosis of acquired heart disease. The cyanosis associated with congenital heart disease is usually present at birth, the so-called blue baby. It is especially likely to be noted when emphasized at the time the baby has a spell of crying. Polycythaemia with as many as eight or ten million red cells per cubic millimetre of blood may occur. Cough, dyspnœa, convulsions, bulging of the precordium, and clubbing of the fingers and toes are common. Many cases show a tendency to epistaxis and to pulmonary tuberculosis. Congenital abnormalities elsewhere in the body may be associated. It should be remembered, however, that any or all of the above symptoms may be absent.

Some of the defects may be mentioned.

**ANOMALIES OF POSITION.**—1. *Dextrocardia* is associated with transposition of all the viscera. Symptoms are absent. The condition is commonly noted in the course of

routine examination, by the location of the apex impulse, percussion dulness, and maximum intensity of the heart sounds in the right chest, in an exactly similar relationship to each other as occurs in the heart normally located to the left of the sternum. Physical examination should demonstrate that the liver and stomach have changed sides respectively. Röntgen examination will confirm the *situs viscerum inversus* of which dextrocardia is but one feature. The electrocardiogram, in a case of congenital dextrocardia, shows an inversion of all waves in lead I, in which it differs from that of a heart displaced to the right by pleural exudates, pneumothorax, tumors, inflammatory processes in the chest, etc. The acquired form may be differentiated by the absence of evidence of associated transposition of the abdominal viscera.

2. *Ectopia cordis*.—The cervical form is found only in the fetus. The heart may be located below the diaphragm. An imperfect closure of the parietes of the chest is sometimes present in the pectoral form of ectopia cordis.

PULMONARY STENOSIS.—This is the most common type of congenital defect in the heart. The stenosis may be at the level of the pulmonic valve or below, at the junction of the conus arteriosus and main cavity of the right ventricle. In many cases the interventricular septum fails to close in its upper part, and in addition the ductus arteriosus remains open. These latter defects serve somewhat to offset the pulmonary stenosis and the patient may have relatively few symptoms, whereas when pulmonary stenosis exists alone, the symptoms are pronounced and the patients rarely live over a year.

The diagnosis is based on evidence of enlargement of

the right ventricle, and a systolic murmur and thrill of maximum intensity over the second left costal cartilage. The murmur may be transmitted to the neck, especially if the ductus arteriosus is patent.

Caution should be used in assessing a systolic murmur and even thrill over the pulmonic area, in the absence of other signs or symptoms of abnormality. This is especially true in young babies. Thayer and MacCallum<sup>1</sup> have shown that it is extremely easy to produce both a murmur and thrill by light pressure on the infundibulum or pulmonary artery. Such a murmur and thrill commonly are much lessened or absent on inspiration. It is not unusual to observe the gradual disappearance of a systolic murmur over the second left interspace. The relationship of the vascular structures to the chest wall and the presence, in the infant, of the thymus gland, are the probable objects which exert external pressure and cause this innocuous murmur.

#### PULMONARY REGURGITATION<sup>2</sup> is rare.

**PATENT FORAMEN OVALE.**—A small opening in the interauricular septum is a common finding at autopsy in the essentially normal heart. In such cases signs and symptoms are absent in life. If a larger defect is present, signs and symptoms are usually present. The latter may be severe, especially when, as is often the case, the patent foramen ovale is associated with other abnormalities. The murmur attributed to non-closure of the foramen ovale may be systolic or both systolic and diastolic. These murmurs are audible over the second, third, and fourth inter-

<sup>1</sup>THAYER, W. S., and MACCALLUM, W. G.: *Amer. Jour. Med. Sci.*, cxxxiii: 249, 1907.

<sup>2</sup>CAUTLEY, E.: *Brit. Jour. Child. Dis.*, London, Oct.-Dec., 1920, 17, Nos. 202-204, 187.

costal spaces and are not transmitted to the neck. Patency of the foramen ovale makes possible the rare "crossed or paradoxical embolism," for example an embolus to the brain from venous thrombosis of the leg.

It seems fallacious to believe that the successful closure of the foramen ovale is in any way dependent upon the position in which the baby sleeps during the first two weeks of life. The valve-like fold or membrane, if present, will be kept in contact with the margin of the foramen by the greater pressure existing in the left auricle after birth. This increase in the left intra-auricular pressure is a natural result of the assumption of the pulmonary circulation.

**PERFORATE INTERVENTRICULAR SEPTUM** is a frequent defect, and, as said above, is often associated with pulmonary stenosis. The signs may resemble those of mitral insufficiency. A loud systolic murmur, not transmitted to the neck, may be heard over the third and fourth interspaces to the left of the sternum. Precordial bulging may be present.

**PATENT DUCTUS ARTERIOSUS** (*ductus Botalli*).—The pulmonary artery is frequently dilated, which may be recognized by dulness in the second left interspace, with a pulsation, thrill, and loud systolic murmur which may reach the vessels of the neck or the fourth dorsal vertebra. Excessive dilatation of the pulmonary artery has been said to have compressed the recurrent laryngeal nerve, with subsequent hoarseness. In contrast to pulmonary stenosis, the pulmonic second sound is usually clear. An interesting point is that the Corrigan pulse and capillary pulsation have been reported<sup>3</sup> as frequently associated with this defect.

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<sup>3</sup> WALKER, R. W. S.: *Heart*, ii, 3:249, 1911.

COARCTATION OF THE AORTA or persistence of the isthmus of the aorta. This is located in the posterior part of the aortic arch, and usually involves the extremity of the fifth left branchial arch, from which the ductus arteriosus is derived. A fibrous cord may connect the arch and the descending aorta, or a complete separation may exist. In such cases, the blood is delivered by the development of a collateral circulation; the upper intercostal arteries from the second part of the subclavian may communicate with the first aortic intercostal, also branches of the thyroid axis and the internal mammary arteries may do the same. This condition may exist in an adult and should be suspected if enlarged and tortuous arteries are detected on the back of the abdomen.

A similar but rare malformation is an obliteration of the aorta just beyond the origin of the left subclavian artery. In this case the ductus arteriosus usually supplies blood to the descending aorta.

AORTIC STENOSIS is uncommon. A systolic murmur and thrill, of maximum intensity at the second right costal cartilage, and transmitted towards the neck, are produced. As other anomalies, such as septal defects and patent ductus arteriosus, are usually associated, there may be other murmurs present, and the heart varies as regards the predominance of left or right ventricle.

TREATMENT.—Congenital cardiac defects cannot be removed. Good hygiene and the avoidance of physical and mental strain are indicated. Treatment is largely symptomatic. Digitalis has but little place. If evidence of cardiac embarrassment is absent, the patient should lead an essentially normal life.

## CHAPTER XV

### EFFORT SYNDROME: IRRITABLE HEART

THE condition designated by the above title appears in the literature under various terms, such as, "irritable heart," "cardiac neurosis," "nervous heart," "neuro-circulatory asthenia," "functional heart disease," etc., and in the army parlance as, "soldier's heart," and "D.A.H." (*i.e.*, disordered action of the heart). Much emphasis was placed upon effort syndrome in the World War, but Jacob M. DaCosta,<sup>1</sup> as a result of observations made on soldiers of the American Civil War, clearly described the condition in 1871 under the designation of the "irritable heart of soldiers." No term as yet seems entirely satisfactory; the double title used above serves to emphasize two of the more common types. The characteristic feature of effort syndrome or irritable heart is that, in the absence of evidence of true cardiac disease, symptoms associated with physical effort occur out of proportion to the slight amount of physical exertion or emotional excitement required to excite them.

**ETIOLOGY.**—Many cases may properly be attributed to recent or still present infections. In some, the condition seems to have a constitutional background, as a lack of good physical development. A sedentary life is a predisposing factor. In others, nervous influences predominate. It has been thought that some degree of hyperthyroidism might be the cause, but careful studies appear to have shown that such is not the case. Physical exercise, in

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<sup>1</sup> DA COSTA, J. M.: *Amer. Jour. Med. Sci.*, lxi: 17, 1871.

excess of the powers of the individual, and psychic strains will produce the symptom complex in the predisposed. The importance of infection in the causation of this disturbance should not be forgotten.

**SYMPTOMS AND SIGNS.<sup>2</sup>**—The chief symptom is rapid heart action usually attended by precordial pain, shortness of breath, dizziness, and oppression. The general appearance varies from healthy to florid or pallid. Tremors of the hands are common. The hands, in some cases, are cold and clammy and may be cyanotic, especially when they are in a dependent position. The pulse tends to be small and compressible. The blood pressure findings are inconstant.

The heart rate is usually somewhat over 100 and is readily increased by exercise or excitement, *i.e.*, it is an unstable tachycardia. Sinus arrhythmia is not uncommon. The respirations are not accelerated in proportion to the pulse rate.

Enlargement of the heart does not occur. On palpation, some degree of hyperesthesia of the precordia is present in nearly all who complain of recent severe pain. At times the latter may radiate down the left arm similarly to angina pectoris. A systolic murmur may often be noted modifying the first heart sound at the apex. In other cases, the first sound is described as split, or shortened, while the pulmonic second sound is often very distinct. None of these findings, it will be seen, are those of true cardiac disease.

When the patient is in bed or kept sufficiently quiet

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<sup>2</sup> If he has not already done so, the reader should read the excellent book by THOMAS LEWIS, *The Soldier's Heart and the Effort Syndrome*, Paul B. Hoeber, New York, 1919.

and free from excitement, the above symptoms usually disappear completely or in a large measure.

**DIAGNOSIS.**—The presence or production of the symptoms and signs normally associated with physical exercise, out of proportion to the exertion to which the patient has been subjected, and the absence of definite evidence of heart disease, should suggest effort syndrome or irritable heart. If the patient is of inferior physique, has incurred some recent infection, or has been subjected to undue physical or emotional strain, the diagnosis becomes increasingly probable. Care should be taken to exclude rheumatic or syphilitic heart disease.

Paroxysmal tachycardia should be differentiated by the fact that it occurs in attacks, with a sudden ending, and the rate remains essentially unchanged regardless of change in posture, etc. It is in no sense an unstable tachycardia.

The heart in hyperthyroidism presents perhaps the most difficult problem in differential diagnosis. Decision should be reserved until the patient has been thoroughly examined and sometimes until the effect of treatment has been observed. Simple rest, or the recumbent posture, will usually quickly lower the pulse rate, whereas in true hyperthyroidism, there is a tendency for some degree of tachycardia to persist even in sleep. When doubt remains, the result of the determination of the rate of the basal metabolism is decisive.

**PROGNOSIS.**—Much depends on the probable cause. If the latter is an infection, its effects may usually be expected to wear away. A large proportion of the patients in whom hyperesthesia of the precordia is present are unable to

perform any but the simplest exercises.<sup>3</sup> Marked breathlessness and very high heart rates tend towards an unfavorable prognosis. If the patient is young and lacking in physical development, graded and systematic exercise may succeed in totally relieving the condition. In the older patients, or in those whose co-operation is less satisfactory, not so much should be expected from physical therapy. Considerable depends in the individual case as to what sort of occupation he is to follow or in what environment he is to live.

Some conception of the prognosis in well-treated cases may be gained from noting the results in the British army. The disposition of these men on discharge from the hospital was as follows:<sup>4</sup> Fit for general service 20 per cent., fit for hardening or labor 30 per cent., fit for light or sedentary work 30 per cent., and permanently unfit 20 per cent. The average length of stay in hospital had been six to ten weeks.

**TREATMENT.**—The indication is clear to treat the patient and not his heart. Search should be made for a focus of infection and, if present, measures should be instituted to free the patient from its influence.

Time should be taken to make it clear to the patient that true heart disease is not present, and to obtain his co-operation in carrying out the treatment. A systematic course of training, starting with exercise sufficiently light and short for the individual patient and gradually increasing, will often be of much benefit. These measures can be employed more satisfactorily, it is true, when patients

<sup>3</sup> MEAKINS, J. C., GUNSON, E. B.: "The Occurrence of Hyperalgesia in the 'Irritable Heart of Soldiers,'" *Heart*, vi: 343, 1917.

<sup>4</sup> LEWIS, THOMAS: The Soldier's Heart and the Effort Syndrome, p. 94.

are handled in groups, as was done in the army, but results can be obtained even with single cases if sufficient time and attention is applied. The patient, in some cases, should be warned that at first he may experience attacks of dizziness or palpitation, and if so he should be neither discouraged nor alarmed, but should rest from one half to two minutes and then try again. In a few days, in successful cases, the tendency to untoward symptoms will lessen, and thus the vigor and duration of the exercise may be increased until the patient works back to a condition of physical fitness.

The patients in whom psychic stimuli are the apparent cause of the symptoms of an irritable heart are to be treated by the measures suitable for neurasthenia. The physician should obtain sufficient acquaintance with the patient's environment and philosophy of life to enable him to point out things that may properly be avoided and the methods by which the patient may achieve greater nervous stability.



SECTION III  
FUNCTIONAL CONDITIONS



## CHAPTER XVI

### FUNCTIONAL CONDITIONS

IN THE preceding chapters, frequent reference has been made to heart failure, angina pectoris, and the various arrhythmias. These conditions will now be presented in the above order. It is emphasized that they should not be considered complete entities; they are merely functional conditions which may be associated with the several types of heart disease.

#### HEART FAILURE

The term "broken compensation" has long been employed for this condition. However, since the explanation of cardiac embarrassment on the basis of mechanical effects has been largely abandoned in favor of infection as a cause, "failure of compensation," with its implication of hypertrophy, seems less desirable. Symptoms of heart failure may appear in a paroxysm of tachycardia in which no hypertrophy has ensued.

Heart failure may be said to be present when there are a certain set of symptoms and signs that are untoward for the patient under examination. The picture is not unlike that which may be produced in large part in a healthy individual when exercise is pushed to the physiological limit and beyond, but it is present in the patient with heart disease while at rest, or as a result of exertion insufficient to call it forth in health.

The first indications are symptoms, such as pain, breathlessness on exertion, and undue fatigue. The tolerance of

exercise is reduced; this statement should be checked by the remarks on this in the chapter on Effort Syndrome and in that on Methods of Examination. Symptoms referable to the stomach are common.

Heart failure with congestion is a familiar type. An important sign is an engorgement of the cervical veins. If the veins are distended when the patient is standing or sitting (care being taken to avoid pressure on the abdomen) it may be confidently stated that the pressure is raised in the veins. The right heart is enlarged, but it may be remarked that the results of percussion, etc., to detect this are equivocal. The evidence obtainable from the condition of the veins in the neck is more trustworthy. As the heart failure undergoes further development, the breathlessness increases, and cyanosis, râles at the pulmonary bases, swelling of the liver, edema of the legs, ascites, and a lessening in the urinary output, appear. As stated elsewhere, serious heart failure may exist without edema. Angina pectoris is a form of heart failure, but it is sufficiently distinctive to demand separate presentation.

The treatment and other details of heart failure are sufficiently considered in other parts of this book.

### ANGINA PECTORIS

The term angina pectoris is applied to a condition characterized by attacks of pain over the heart, with a tendency to radiate to the left shoulder and arm, and often accompanied by a sense of impending death.

**ETIOLOGY.**—Males are more frequently affected. Certain families are known to be prone to this affection. Angina pectoris may occur at any age after infancy, but it is much more common in patients forty-five to fifty years

of age or more. Excessive wear and tear of life are apparent factors. Increased weight, in the causation of angina pectoris, has of late been given to infectious diseases, especially syphilis. Next in order comes influenza, then rheumatic fever, infections with the streptococcus, and occasionally other infections. The commonest conditions associated with angina pectoris are arteriosclerosis, syphilis, and chronic hypertension.

Authorities are not agreed as to the pathological cause of angina pectoris. The view which perhaps has gained the widest acceptance is that the disease could be traced to sclerosis of the coronary arteries or to a spasm of these vessels causing an ischemia of the myocardium. More recently, however, an aortitis as the true lesion has gained many advocates. In view of the interest of the subject it seems proper to discuss briefly these leading theories.

The coronary sclerosis theory does not explain the cases, now numerous, in which post-mortem examination has disclosed normal coronary arteries. Also, doubt is thrown upon the importance of sclerosis of the coronary arteries since, in the age period in which angina pectoris is most frequent, arteriosclerosis is also common. In fact, in necropsies conducted on elderly subjects, it is the rule to find some degree of coronary sclerosis associated with more general arteriosclerosis, and yet but comparatively few of these patients in life were afflicted with angina pectoris. The coronary arteries may be sclerosed almost to obliteration without apparent damage to the myocardium, or symptoms during life. This is probably explainable by the establishment, provided the silting up of the coronary channels is slow enough, of compensatory circulation, by

means of the free anastomosis of the right and left coronaries. Also, Pratt<sup>1</sup> has pointed out that the veins of Thebesius may supply blood sufficient to maintain the integrity of the myocardium.

If angina pectoris is not due to coronary sclerosis, then some say it is caused by attacks of claudication causing myocardial ischemia. As yet I am unaware of any data in proof of this conception. The accuracy of the analogy to claudication in the horse is open to question, as the horse so attacked limps, but it is not so with the heart during an anginal attack. The weight of evidence from the physiological laboratory makes it doubtful if a contraction or spasm of the coronary arteries ever takes place.

Sir Clifford Allbutt<sup>2</sup> writes very compellingly in favor of the aortitis theory of angina pectoris. In brief, the condition is presumed to be due to irritation of the nerve end-plates in the first part of the aorta by the placques of an aortitis, and is especially liable to occur in conditions causing a rise of the intra-arterial pressure and subsequent stretching of the investing membranes of the root of the aorta. There may be a partial analogy to the pain caused by dragging on the mesentery. To quote from this distinguished author: "Still, so far as I am aware, there is not a case of angina on record, on responsible authority, in which disease of the coronaries was the sole lesion; none, that is, in which, for instance, the state of the suprasigmoid aorta also had been histologically examined, within and without, and declared normal."

While the advocates of the aortic causation of angina

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<sup>1</sup> PRATT, F. H.: *Amer. Jour. Physiol.*, 1: 86, 1898.

<sup>2</sup> Diseases of the Arteries, Including Angina Pectoris, Macmillan & Co., Ltd., 1915.

pectoris deny the coronary theory, they do believe that the condition of the coronary arteries and the myocardium is of importance in determining the survival or death of the patient experiencing the anginal attack. Thus, death is explained by reflex vagus inhibition of the heart, and failure, in cases with poor myocardium, of the process of ventricular escape. It seems more likely, however, that death is due to the occurrence of fibrillation of the ventricles.

Mackenzie writes<sup>3</sup> that he is uncertain of the exact cause of angina pectoris but considers some form of heart exhaustion, and treats it on that basis. In the opinion of the author, this is a wise and conservative position.

An appreciation of the factors involved makes apparent the difficulties in the way of obtaining the data necessary to establish the etiology of angina pectoris. As yet the verdict must be "not proven," and yet the above theories serve as important leads from which to base our therapeutic measures.

**SYMPTOMS.**—Typical angina pectoris is characterized by paroxysmal attacks of pain over the sternum, often radiating to the left shoulder and arm, and commonly associated with a sense of impending death.

The pain varies from a mere sense of uneasiness or constriction to that of intense agony. Its location is under or near the sternum, particularly the upper portion. Allbutt points out that if the pain is precordial or near the nipple it is not due to angina pectoris but to some other affection of the heart, usually with failure.

The pain in angina shows a distinct tendency to radiate to the left shoulder and arm. A favorite location is over

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<sup>3</sup> MACKENZIE, JAMES: *Oxford Medicine*, ii, 387, 1920.

the area of the lesser internal cutaneous nerve. It is less common for the radiation to extend below the elbow, and when it does so, it is more likely to follow the ulnar than the median distribution. Radiation to the right upper extremity and to the neck occurs, but is less common. At times the pain may strike through to the region near the inferior angle of the left scapula or to a larger area in the back. Rarely the pain may be felt in the lower jaw or molar tooth. Epigastric and abdominal locations are occasionally described as occurring in angina pectoris, and have even been accompanied by jaundice.

At times the pain may be absent over the sternum and present in one of the points of radiation. Thus patients may present themselves with the complaint of pain only in the arm or hand. Later on, these cases may develop typical angina pectoris.

The attacks are commonly brought on by exertion, as by climbing an ascent or walking against the wind. Large meals may precipitate an attack; in fact, it is felt that many of the attacks and deaths from so-called "acute indigestion" are really due to angina pectoris. Sudden chilling of the body surface, as a cold shower or cold sheets, may induce a paroxysm. The discomfort or irritation of a distended bladder, due to prostatic obstruction, may provoke the paroxysm. The attacks usually occur during the waking hours, but occasionally may disturb the patient at night. Victims of angina pectoris are also prone to develop an attack following an emotional state, such as sudden rage. Thus, there is some foundation for the novelist's tendency to dispose of an elderly character by sudden death after intense rage or joy.

An indescribable sense of impending death often accompanies an attack of angina. If present, it is of considerable diagnostic value. Often the patient will spontaneously mention this feeling, in more reticent individuals it may be elicited by suitable questioning.

When seized by an attack of angina, the victim practically always remains motionless. The patient, who may be walking at the time, will pause to look in a window or lean against a fence, and resume his course only after the attack has ceased. The respiration is never deepened, but is often shallow or temporarily suspended. It is common for the skin to pale markedly, and beads of perspiration may appear on the forehead.

The pulse, to one's surprise, may show practically no change. In fact, unless some other cardiac affection is present, no change will be noted in the pulse beyond an occasional elevation of the rate by a few beats. There is a slight divergence of opinion as regards blood pressure, but in the experience of the author, it shows no alteration in the peripheral pressure.

Death may suddenly terminate the attack.

**DIAGNOSIS.**—Angina pectoris, it will be seen, is to be diagnosed from the history and symptoms, and not from physical signs. In well-marked cases, the agonizing pain, with its tendency to radiation, and accompanied by a sense of impending dissolution, clearly points to angina pectoris. But it is in the less-developed cases, where perhaps the pain is felt only at one of the points of radiation, or in the case in which the only symptom is an indescribable feeling of uneasiness over the sternum, that care should be taken not to treat the matter too lightly. Questions should be

directed to ascertain the circumstances under which these symptoms occurred, and, if there is evidence of syphilis or if the patient is beyond middle age, the possibility of angina pectoris should be considered. It should be mentioned here that angina pectoris appears in both severe and mild forms. Thus, if the symptoms are light, one is nevertheless dealing with the same disease as if the patient were experiencing intense agony.

The presence of other cardiac affections in which pain is a symptom may complicate the picture, especially if evidence of cardiac failure has appeared. But if the observer keeps clearly in mind the symptoms discussed above, he may yet be able to diagnose both conditions.

True angina pectoris is to be distinguished from mock angina. The latter is held to be a vaso-vagal attack which, unlike true angina, is spontaneous in origin. Huchard's well-known table (which I have modified somewhat) emphasizes some of the contrasts.

Allbutt writes of mock angina, as contrasted to true angina: "The pain is different, the pulse is different, the panting is different, the behavior is different, the storm is different, the duration is different, the causes are different, the issue is different."

The use of tobacco in excess may rarely cause mock angina.

Occasionally thrombosis or embolism of the coronary circulation is to be differentiated. An acute pain may occur, but, unlike true angina, it may persist without abatement for twenty-four to thirty-six hours. It is commonly associated with a marked fall of blood pressure, dyspnoea, and pulmonary edema. Sir William Osler, and others,

have pointed out, however, that even in cases of sudden death due to blocking of one of the coronaries, or a large branch such as the anterior, the seizure is usually painless. This fact and the marked fall in blood pressure, which

#### ANGINA PECTORIS

Most common in men between forty and fifty.

Paroxysm induced by exertion, etc.: diurnal; few in number.

Pain intense, of short duration, substernal; sensation of cardiac compression.

Patient inarticulate; immobile attitude; respiration shallow.

Pulse unchanged.

Nervous symptoms absent.

Prognosis grave, often fatal.

An aortitis, or stenosis of coronary arteries often present.

#### MOCK ANGINA

Commonest in women, at any age.

Spontaneous; often nocturnal and periodic; frequent.

Less severe, lasting hours; epigastric; sensation of distention.

Agitation, and activity; respiration often accelerated.

Pulse often weakened.

Neurasthenic or hysterical stigmata.

Never fatal.

No lesions found.

also follows experimental ligation of the coronaries, appears to throw further doubt on the theory of an ischemia due to coronary claudication as the explanation of angina pectoris.

Alteration in the ventricular complex in the electrocardiogram may be observed.<sup>4</sup>

<sup>4</sup> PARDEE, H. E. B.: *Arch. Int. Med.*, 26: 244 (Aug., 1920).

PROGNOSIS.—The prognosis should be guarded, as death is possible in the first or any subsequent attack. If heart failure ensues, the anginal attacks usually cease, but may reappear with improvement in the heart action. The late John H. Musser, Sr., emphasized that a change of physical type occurred in those patients in whom the appearance of cardiac failure was associated with the disappearance of the anginal attacks. That is, the vigorous, active individual soon took on the characteristics of the broken-down subject.

If the angina is due to syphilis, considerable relief or even an arrest of the disease may result from timely treatment. Angina due to an infection other than syphilis tends to subside spontaneously.

TREATMENT.—The treatment is directed towards the relief of the attack and the prevention of their repetition.

For the attack, a rapidly acting vasodilator has been found most helpful. The inhalation of the fumes of a "pearl," containing 5 min. (0.3 c.c.) of amyl nitrite, freshly crushed in the handkerchief, gives quickest relief. Nitroglycerine, however, is generally the more satisfactory remedy and is preferred by most of the patients. A tablet, gm. 0.006 ( $\frac{1}{100}$  gr.), allowed to dissolve under the tongue, should act in about three to five minutes. The same amount may be repeated in ten minutes if relief has not been obtained. In some individuals, it may be necessary to work up to a much larger dosage.

Morphine, gm. 0.01 to 0.015 ( $\frac{1}{60}$  to  $\frac{1}{4}$  gr.), is a sovereign remedy in a severe attack and has the added benefit of ensuring a quiet patient for several hours.

Heat, in the form of a hot-water bag, electric pad, etc.,

may be applied to the precordium. The patient ought properly to be kept at rest for several days, as it has been noted that there is a tendency for attacks to occur in cycles, and the attack may be repeated.

As it is only occasionally that the physician is present at the time of the attack, the patient or a member of the family should be carefully instructed in the treatment of the acute attack. If the physician or other responsible person is present during the paroxysm, the nitroglycerine or morphine may be given hypodermically.

Between attacks, attention should be paid, primarily, to therapy based on a study of the case from an etiological viewpoint. It is especially important that antisyphilitic treatment be given whenever indicated, and some would say it should be given a trial in each case. If it is decided that arteriosclerosis is the causative factor, the treatment pertaining to that disease is indicated. Occasionally the underlying condition is hypertensive heart disease, for which appropriate therapy should be instituted.

The conditions predisposing to attacks of angina pectoris should be explained to the patient, so that they may, if possible, be avoided. These are: exertion, excitement, worry, chilling, sexual intercourse, constipation, dyspepsia, late suppers, large meals, and foods favoring flatulency. If there is a tendency to gastric flatulency, the patient should be provided with some aromatic, as Hoffman's anodyne (*Spiritus Ætheris Compositus*), or some mixture as:

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*Spiritus ammoniae aromatici*

*Spiritus chloroformis*

*Spiritus ætheris compositi* āā, 3 i ss.

*Sig.:* To be taken in hot water.

In elderly men, with hypertrophied prostates, measures should be taken to avoid bladder distention.

The use of nitroglycerine 0.006 gm. ( $^1/_{100}$  gr.) three times a day, as sometimes prescribed, is problematical, as the action of the drug is less than one hour in duration. If a more continuous action of a vasodilator is desired, nitroglycerine should be repeated at frequent intervals, or sodium nitrite 0.06 gm. (1 gr.), or erythrol tetranitrate 0.03 to 0.06 gm. ( $^1/_{2}$  to 1 gr.) every three to four hours, should be substituted. It has often been proven of value, however, to advise the patient to take his dose of nitroglycerine just before any known physical exertion, as a walk to the store, etc., in order to ward off an anginal attack.

Good results have also been obtained from the swallowing of the nitroglycerine already in solution. It should be remembered that, to be active, the drug should be fresh, and it is, therefore, a wise precaution for the patient to renew weekly the emergency supply of nitroglycerine that is carried on his person. For those who hesitate to prescribe nitroglycerine, due to uncertainty as to its toleration by the patient, it is suggested that gr.  $^1/_{100}$  of the drug be administered as a test while the patient is yet in the office.

## CHAPTER XVII

### THE ARRHYTHMIAS

A LARGE part in the recent advance in the subject of heart affections has come from the studies of normal and abnormal cardiac function. The use of graphic methods of examination has contributed greatly, with but few exceptions, to the elucidation of the nature of irregular or disordered mechanism of the heart. Now that the essential facts have been established, it is found that the arrhythmias may often be correctly diagnosed without resort to the use of the polygraph or electrocardiograph. Since the contributions of the polygram and the electrocardiogram have been described, briefly it is true, in earlier chapters, in the following, the chief features of the various arrhythmias will be presented from the standpoint of observations made by the more usual clinical methods. Attention is drawn to the fact that the arrhythmias are not true entities in the sense that are rheumatic heart disease, cardiovascular syphilis, etc.; these functional conditions may be features of various heart affections.

#### SINUS ARRHYTHMIA

In sinus arrhythmia the impulse from the pacemaker of the heart (*i.e.*, the sino-auricular node) starts irregularly and causes thereby an arrhythmia of the whole heart.

It is due to variation in the control of the heart by the vagus nerve. The usual type is that in which the heart accelerates with inspiration and slows with expiration, but the conditions may be reversed. A much less frequent

form of sinus arrhythmia is phasic irregularity, in which the whole heart slows periodically and not in relation to respiration. Respiratory irregularity of the heart rhythm is present in almost all children. After the age of puberty, it rapidly becomes less common, and in adults is inconspicuous or absent save in forced breathing. Respiratory variations in the pulse rate are often conspicuous during convalescence from acute illnesses.

Sinus arrhythmia causes no symptoms. It is usually detected solely as the result of physical examination. In most cases it may be considered physiological. If it has appeared in an adult during convalescence from some febrile disease, it will often diminish or disappear in a few weeks or months.

No treatment is needed. It may be temporarily abolished by the action of atropine.

It is important that the nature of sinus arrhythmia be understood, as it may cause unnecessary alarm on the part of physician and patient.

### SINO-AURICULAR BLOCK

Sino-auricular block may resemble the form of heart-block showing dropped beats. Its presence should be suspected in patients in whom, especially after exercise, the heart rate slows gradually to 80 or 70 and then drops to about one half. It may result from digitalis administration; in some cases it is associated with respiration. It appears closely related to sinus arrhythmia and to the action of the vagus nerve. Sino-auricular block is often associated with auriculo-ventricular block. The hypothesis of a lesion or condition causing a block between the

sinus node and the auricular tissue lacks anatomical or physiological proof.

Sino-auricular block is commonly symptomless. Its presence does not imply disease. When due to digitalis, it will disappear on the discontinuance of the drug. No treatment is required.

This arrhythmia is relatively uncommon and is rarely diagnosed save as a result of examination by the graphic methods.

## PREMATURE BEATS OR EXTRASYSTOLES

Premature beats or extrasystoles are very common; in fact, their prevalence is so great that it is suggested that all patients are subject to them at some time. They may arise in the auricle or in the ventricle, being five times as frequent in the latter. Auricular premature beats are, however, more important, as they are often associated with paroxysmal tachycardia.

**ETIOLOGY.**—The causation of extrasystoles is obscure. They are premature beats of the heart in response to stimuli, probably newly generated, and not forming part of the rhythmic series of contractions. Their point of origin, save for rare exceptions, is in some part of the myocardium other than the sino-auricular node. It is commonly held that they are evidence of increased excitability of the cardiac tissues, but according to Lewis,<sup>1</sup> that is pure hypothesis. Premature beats may at times be associated with the action of certain poisons, such as: chloroform, digitalis, strophanthin, aconitine, barium chloride, potassium salts, nicotine, theophyllin, muscarine, and physostigmine,

<sup>1</sup> LEWIS, THOMAS: The Mechanism and Graphic Registration of the Heart Beat, Paul B. Hoeber, N. Y., 1920, p. 342.

tea, coffee; and with certain conditions, as: toxemia, nervousness, arteriosclerosis, syphilis, and hypertension.

While their incidence is most frequent in patients in whom there is structural disease of the heart, their prevalence amongst apparently healthy people largely destroys the significance of this relation.

SYMPTOMS AND SIGNS are often absent. Occasionally the patient is conscious of the cardiac irregularity and states that it feels as though the heart "turned over." Palpitation may be present. Breathlessness on exertion may be noted in the occasional patients exhibiting extrasystoles at a high rate of the heart.

The heart rate is usually under 100, and the premature beats will disappear if the rate is accelerated to over 110. Rare exceptions to the above statement occur; the writer has electrocardiograms showing frequent auricular premature beats even at the cardiac rate of 146 per minute.

On auscultation of a typical case, it is easy to detect the prematurity of the extrasystoles and the succeeding pause. One or two sounds will be audible, according as the extrasystole is strong enough to open the semilunar valves (second sound results from their closure). Whether the extrasystole will be represented in the radial pulse depends upon the amount of blood expelled by the heart. A series of rhythmic heart sounds, interrupted by one that is premature, should at once suggest an extrasystole, but when the irregular beats are frequent, it may be necessary to resort to some method to accelerate the heart rate (unless it is already elevated), and the irregular beats should diminish or disappear.

If the pause following the premature beat is not fully

compensatory, it may confidently be asserted that the beat was of auricular origin,<sup>2</sup> while if a fully compensatory pause is present, the ectopic beat almost certainly comes from the ventricular tissue (with rare exceptions).

**DIFFERENTIAL DIAGNOSIS.**—Premature beats are to be distinguished from fibrillation of the auricles. This is usually evident at once. A rule that has rare exceptions is, that premature beats diminish or disappear following acceleration of the heart rate, as by exercise. When the arrhythmia is due to extrasystoles, the irregularity is not absolute; the prematurely occurring beats are nearly always followed by a pause of increased length. In fibrillation, the post-systolic pauses are unrelated to the time of occurrence of the beats. The ventricular type of venous pulse may be observed in fibrillation, but not in extrasystolic arrhythmia.

Incomplete heart-block, causing dropped beats, is readily confused with premature beats, due to the fact that the latter are often not transmitted to the wrist. The desirability of avoiding this mistake is obvious, since the two conditions differ widely in prognosis and treatment. Extrasystoles are common and dropped beats of relatively rare occurrence. It is unsafe to diagnose a dropped beat, and so heart-block, from evidence obtained solely by palpation of the pulse. This latter diagnosis ought properly to be confirmed by the use of the graphic methods of examination. On auscultation at the apex, the extrasystole may show two heart sounds, but in case merely a single faint sound is audible, this must be differentiated from the occasionally faintly audible sound of the auricle, the contraction

<sup>2</sup> Rare exception: idio-ventricular rhythm interrupted by ventricular extrasystoles.

of which now occurs during the period when the heart should be silent. More often the condition of dropped beat is represented on auscultation by a gap in the heart sounds. The absence of symptoms of heart failure and the prompt disappearance of the irregularity, on acceleration of the heart rate by exercise, are strongly in favor of the simpler diagnosis, namely premature beats. However, after acceleration of the pulse by exercise, the irregularity, due to either premature beats or the dropped beats of heart-block, may completely disappear, and then, as urged by Lewis,<sup>3</sup> particular attention should be directed to the first irregularity that appears as the pulse rate falls. If the arrhythmia is due to premature beats, a premature beat will be heard, while there will be a gap if heart-block is the cause.

**PROGNOSIS.**—The presence of premature beats without other evidence of cardiac abnormality is of little prognostic import. They may come and go, or persist for years, with no evidence of physical impairment.

**TREATMENT.**—The detection of the premature beats should indicate the need of investigation sufficient to discover the presence or absence of evidence of heart disease, and any of the conditions, mentioned above, which have been found in association with this arrhythmia. These associated conditions, or apparent causes, should be removed if possible. Other than this, most cases affected by extrasystoles require no treatment.

When symptoms are present, and especially in the occasional patient in whom the premature beats persist at the higher cardiac rate, the temporary use of bromides and veronal may be of assistance.

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<sup>3</sup> LEWIS, T.: The Soldier's Heart and the Effort Syndrome: Paul B. Hoeber, New York, 1919, p. 72.

## PAROXYSMAL TACHYCARDIA

This is definable as tachycardia of regular rate, and sudden onset and cessation. It is of auricular origin about twenty times as frequently as it is ventricular. This description will be limited to the auricular type.

**ETIOLOGY.**—Paroxysmal tachycardia has been noted as early as the age of six, and as late as seventy-four years; the period of twenty to forty is the commonest. It occurs in females and males in about the proportion of three to two.

The apparent causes are essentially the same as in the case of premature beats (which see). Those who accept the hypothesis that extrasystoles are due to increased excitability of the heart consider that paroxysmal tachycardia indicates a somewhat higher degree of excitability.

The mechanism is obscure. Evidence obtained by the graphic methods of examination discloses that a paroxysm is introduced by a premature contraction, and apparently is a series of the latter repeated in rapid and regular succession. What is not clear, is, how it comes about that these new impulse centres, which commonly have the power to build up a stimulus at a relatively low rate (usually below 100), can suddenly produce a series in rapid succession. Now that it has been established that auricular flutter (which see) is due to circus movement, the hypothesis of a similar mechanism, leading to the production of paroxysmal tachycardia, is very tempting.

**PATHOLOGY.**—This is unknown in mild cases, as they are not examined; in some instances myocardial changes have been found.

**SYMPTOMS AND SIGNS.**—Exertion or excitement usu-

ally start the paroxysm, which rarely persists for more than a few hours, though it may continue for several weeks. A duration of a few minutes or seconds is not uncommon; exceptionally long and short attacks may occur in the same patient. The condition is felt by the patient as palpitation. There may be dyspnoea and some precordial or epigastric distress. The sensation of pounding on top of the head is at times most unpleasant. In the paroxysms of long duration, the liver may swell and all the signs of cardiac failure be in evidence.

The pulse rate is commonly over 140 to the minute and under 200, which figure is arbitrarily set to separate paroxysmal tachycardia from flutter of the auricles. A striking feature is the regularity of the rate, and that it is hardly affected by change of posture, though Lewis<sup>4</sup> has reported a variation as high as twelve beats between the rate on standing and on lying. In addition to the rapid rate, the volume of the pulse is often small and subject to considerable respiratory change. The size of the heart during a paroxysm was diminished when examined radioscopically by Vaquez and Bordet;<sup>5</sup> in a persistent attack, dilatation of the heart may ensue.

**DIAGNOSIS.**—This is usually evident from the presence of a regular tachycardia, less than 200 to the minute, and but slightly affected by change of posture. The use of graphic methods is helpful on the doubtful cases.

It is more often the case, however, that the patient is seen between attacks, and then the diagnosis must perforce

<sup>4</sup> LEWIS, T.: The Mechanism and Graphic Registration of the Heart Beat, Paul B. Hoeber, New York, 1920, p. 244.

<sup>5</sup> VAQUEZ, H., and BORDET, E.: Le Cœur et l'Aorte, J. B. Bailliere et Fils, Paris, 1920, p. 155.

rest upon the history. Of particular value is a positive history of an abrupt beginning and ending of the attacks, the rate and regularity of the heart during the "palpitation," and the association of vomiting with the ending of an attack, or the details of any of the measures by which the patient may have learned to terminate the paroxysm.

The tachycardias due to hyperthyroidism and to the effort syndrome or irritable heart should be differentiated by the fact that they in no sense possess the remarkable regularity in rate, or the abrupt onset and cessation, so characteristic of paroxysmal tachycardia.

The distinction from auricular flutter is based upon the tendency of the latter to show a greater degree of heart failure and to be permanent. An electrocardiogram should settle the doubt. The matter will be further discussed under auricular flutter.

**PROGNOSIS.**—This is usually good in the short and early attacks. If the paroxysm is of the persistent type, occasionally grave heart failure and death may result. The prognosis is influenced, of course, by evidence of other abnormality of the heart.

**TREATMENT.**—The attack may be promptly terminated, in a considerable portion of the cases, by resort to procedures which produce stimulation of the vagus nerve. The latter is accessible in the neck where the right and left trunks course downward in the posterior part of the respective carotid sheaths, and may be stimulated by compressing the artery, at the level of the cricoid cartilage, between the ball of the thumb and the vertebral column. The pressure should be sufficient to obliterate the carotid pulsations, and should be continued 15 to 20 seconds. This manœuvre

should be performed first in the right neck, and, if the tachycardia persists, should then be carried out over the left carotid. Support of the patient's neck, from behind, by the opposite hand, is often helpful. Sometimes success comes only after repeating, two or three times, the above method of stimulation of the vagus. Untoward results, *i.e.*, convulsions from cerebral anemia, have sometimes followed the simultaneous compression of both vagi.

If the paroxysm is uninfluenced by pressure on the vagus in the neck, the nerve may be stimulated by pressure for a similar duration on the eyeballs, through the closed lids. Since this procedure readily causes pain to conscious patients (some cases occur while under surgical anesthesia), not more than moderate pressure can usually be tolerated.

A change in the position of the patient, such as bending well over, has helped. Drinking ice-cold water has relieved some patients. Deep breathing, and the holding of a deep breath, may be tried. The induction of vomiting by ipecac, or by inserting the finger in the throat, has terminated many attacks. An abdominal belt, or firm pressure on the abdomen, has proven successful at times, perhaps due to flushing of the heart by the return of an extra amount of venous blood.

If the attack continues, an ice bag to the precordia is indicated. Digitalis should be employed in persistent cases, but one should not rush to this treatment.

Between the attacks, the important measures are, first, rest, and then to avoid the apparent exciting cause, as excitement, exertion, and heavy meals, etc. Bromides and veronal may be of service where nervous factors are prominent.

## AURICULAR FLUTTER

This may be defined as a condition in which the auricle beats regularly at a rate averaging about 300 per minute. It is a relatively rare cardiac rhythm.

**ETIOLOGY.**—Auricular flutter may be present as early as the age of five, and in advanced years; the most frequent period is forty to sixty years. Among the conditions associated with flutter are arteriosclerosis, coronary sclerosis, rheumatic heart disease, and syphilis. Nervousness appears to be much less of a factor than it is in paroxysmal tachycardia.

The mechanism of flutter of the auricles would seem to have been established in 1920, by Lewis and his collaborators.<sup>6</sup> These authors demonstrated, in experimentally produced flutter, the continuous propagation of a contraction wave round and round the ring of tissue surrounding the junction of the venæ cavae with the right auricle. This confirms the suggestion of Mines,<sup>7</sup> that circus motion could occur in the human heart. Mines cut a ring of tissue from the auricle of a large ray fish and demonstrated the phenomenon of circus motion thereon. He writes, "In such a preparation, a single stimulus, applied to any point in the ring, starts a wave in each direction. The waves meet on the opposite side of the ring and die out. But by the application of several stimuli in succession, it is sometimes possible to start a wave in one direction while the tissue on the other side of the point stimulated is still refractory. Such a wave runs round the ring sufficiently slowly for the refractory phase to have passed off in each part of the ring

<sup>6</sup> LEWIS, FEIL, and STROUD: *Heart*, 1920, vii. The observations have been extensively amplified by Lewis and co-workers in a series of papers in *Heart*, viii, 1921.

<sup>7</sup> MINES, G. A.: *Jour. Physiol.*, xlvi: 349, 1913.

when the wave approaches it. Thus, the wave circulates and may continue to do so for fifty revolutions or more.<sup>28</sup> The contraction wave, circulating in this central ring, sends contractions centrifugally off over the auricle, just as waves spread from a stone thrown into a pond. The rate at which these impulses (steadily propagated round the anatomical ring mentioned above) recur is so much more frequent than that of the sino-auricular node, that the rapid rhythm of the flutter dominates the heart.

**SYMPTOMS AND SIGNS.**—The rhythm is generally constant. The symptoms are palpitation and, usually, those of cardiac embarrassment. The onset is sudden and the cessation gradual, usually as a result of treatment. The rapid auricular contractions are sometimes faintly audible. The ventricle rarely beats at the rapid rate of the auricle; in fact, some degree of heart-block is usually present. Therefore, the ventricular and the pulse rates may give but small clue to the extreme frequency of the auricular contractions. An irregularity may be present due to a shifting from 2 to 1, 4 to 1, etc., relation between the auricular and ventricular rhythms.

**DIAGNOSIS.**—The use of graphic methods of examination, and more especially the electrocardiogram, are essential to an accurate diagnosis of flutter of the auricles.<sup>9</sup> Paroxysmal tachycardia differs in that it does not tend to the permanency that is a feature of flutter. The abrupt ending, and the response to vagus stimulation, so often noted in paroxysmal tachycardia, are not characteristic of

<sup>8</sup> MINES, G. A.: *Jour. Physiol.*, xlvi: 349, 1913.

<sup>9</sup> LEWIS, THOMAS: *The Mechanism and Graphic Registration of the Heart Beat*, Paul B. Hoeber, New York, 1920.

auricular flutter. The pulse is always regular in paroxysmal tachycardia, but not necessarily so in flutter.

Simple heart-block may be in question when the ventricular rate is irregular. Success in auscultating the auricular heart sound may disclose if the frequency of the systoles of the auricle is consistent with the rhythm of flutter, or of the slower mechanism that obtains in uncomplicated heart-block. With care, conclusions may, in some instances, be drawn from observation of the jugular pulse. The employment of the graphic methods will settle the doubt.

**PROGNOSIS.**—Flutter of the auricles has been known to persist for six years;<sup>10</sup> it may be transient. It commonly is indicative of serious impairment of the heart, and the expectation of life is shortened.

**TREATMENT.**—This consists in the administration of digitalis to the point of increasing the grade of heart-block, some degree of which is usually present, and in many cases the auricular flutter will change into that of auricular fibrillation, and then on stopping the digitalis, normal rhythm will follow. This latter happy event, unfortunately, does not always ensue, but in these cases the digitalis therapy is of value in that, by its power to increase the grade of heart-block, the ventricular rate may be kept down and cardiac failure averted. Quinidine sulphate (see chapter on treatment) will probably become a useful drug in the treatment of flutter. Rest in bed, and the administration of diuretics, if edema is present, are, of course, indicated.

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<sup>10</sup> LEWIS, FEIL, and STROUD: *Heart*, vii, 1920.

## AURICULAR FIBRILLATION

Auricular fibrillation has been defined by Lewis as, "A condition of the auricle in which some part of its muscle is constantly contracting, but in which the movement as a whole is more or less incoördinate, and therefore ineffectual."<sup>11</sup> It is a frequent and important cause of irregular heart action.

**ETIOLOGY.**—Fibrillation of the auricles may occur in early life and in old age, but it is most common after the age of twenty to twenty-five. It is stated that 25 to 50 per cent. of the cases of fibrillation occur in patients with mitral stenosis, *i.e.*, rheumatic heart disease, and after forty-five to fifty years of age, there appears another large wave of fibrillation cases in those affected with arteriosclerosis.

Many victims of hyperthyroidism, who have been untreated, or in whom treatment has been unsuccessful, are affected later on by fibrillation. This type of arrhythmia may occur in pneumonia. It has been reported in diphtheria, and in syphilis. The frequency of its incidence in syphilis is uncertain, but it seems clear that the impression that fibrillation does not occur in the luetic heart is erroneous; the writer has observed fibrillation in undoubtedly syphilitic hearts, and its association has been reported in the literature. On this point it seems fair to state that in many instances the spirochetal causation of heart disease is frequently unrecognized.

Fibrillation has been produced experimentally by the intravenous injection of poisons, notable amongst which are chloroform, digitalis, strophanthin, adrenalin, nicotine, potassium salts, barium chloride, and aconitine.

<sup>11</sup> LEWIS, THOMAS: *The Mechanism and Graphic Registration of the Heart Beat*, Paul B. Hoeber, New York, 1920.

The mechanism of auricular fibrillation is closely related to that already described for auricular flutter, namely, circus movement. Lewis and his co-workers<sup>12</sup> have proven that fibrillation results from circus movement, but the contraction wave pursues a sinuous path, both in outlying parts of the auricle, and in the path of the central wave in the ring round the junction of the venæ cavæ with the heart. The sinuous course of the wave is due to the fact that it is traveling in muscle that has not fully recovered from the previous contraction and is still partially refractory.

Every contraction of the auricle sends a stimulus to contract down to the bundle of His and thence to the ventricle, and in accordance with this physiological fact it can be seen that in auricular fibrillation, in which some part of the auricle is almost constantly contracting, the auriculo-ventricular conduction tissues are being bombarded by a shower of impulses. Many of these may be conducted to the ventricle, and the latter contracts in response to as many as it is able. Since these stimuli vary in their strength and time of arrival, the rhythm of the ventricle is very irregular. The irregularity in the time of the ventricular contractions accounts directly for the variation in the amount of the content of the blood which can be expelled with each systole. Thus, many contractions may occur so early that the ventricle has not had sufficient time to fill properly, and the ventricular output may be too small to send a wave into the peripheral arteries or even to open the semilunar valves. From this it can be seen that fibrilla-

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<sup>12</sup> LEWIS, FEIL, and STROUD: *Heart*, vii, 1920.

tion of the auricles produces an inefficient rhythm of the heart.

**PATHOLOGY.**—The type of lesion found varies, but scattered foci of inflammation and degeneration are often present in the auricular tissues, and most marked in the specialized tissues. Other pathology consistent with the associated heart disease, as rheumatic or arteriosclerotic lesions, may also be present.

**SYMPTOMS AND SIGNS.**—In brief, these are an irregular rhythm of the heart, and some degree of cardiac failure. The irregularity is termed absolute arrhythmia, which is helpful in emphasizing a difference of fibrillation from all other cardiac arrhythmias. A rule that is rarely broken is that if the heart rate is 120 or more and the rhythm is irregular, one is almost certainly dealing with a case of auricular fibrillation. Exercise tends to increase the irregularity, which again is a distinguishing point.

The pulse varies in volume and in rate. In patients resting quietly in bed, the heart rate may not be more than moderately elevated. If the rate is elevated, it is common to find that not all the beats heard by auscultation of the heart are represented at the wrist. The numerical difference between the rate recorded at the apex and at the wrist is often termed "pulse deficit." Some writers prefer to label "abortive" those heart beats which fail to reach the peripheral arteries. Unlike extrasystoles, the pause following the heart beats which are heard obviously earlier than the average of their fellows bears no relation to the prematurity of the individual beat.

The ventricular type of jugular pulse is present and is often helpful in diagnosis. When the heart rate is rapid,

it is usually difficult or impossible for the observer to analyze the rapid succession of waves, but in most cases there are occasional pauses of sufficient length to permit the disappearance of all waves in the cervical veins. Then attention should be directed to noting if the succeeding heart sound, heard through the stethoscope over the apex, is preceded by a wave in the jugular vein. This will be the "A" wave, described under the polygram, and it does not occur in auricular fibrillation. This observation may be assisted if care is taken that the patient is reclining at an angle that permits the maximum pulsation in the cervical veins. The observer should endeavor to see the silhouette of the pulsating right jugular against the background of the pillow or of a dark cloth. The eye may be aided by placing the edge of a card, or suitable substitute, parallel to the vein and then observing the alteration in the slit between them. Too much should not be expected of the above visual analysis of the venous pulse on the first attempt, but the writer believes it will often be found of value if practised sufficiently to be well done, and if occasionally checked up by the polygram or electrocardiogram.

**DIAGNOSIS.**—The presence of an absolute type of arrhythmia, usually associated with some degree of heart failure, is the most valuable clinical sign. Irregularity of the heart rhythm at the rate of 120 or more, or an increase of the arrhythmia following acceleration of a lower rate, by exercise, save for rare exceptions, indicates the presence of fibrillation of the auricles. The detection, by inspection, of the ventricular form of venous pulse, provided the observer is satisfied with the accuracy of his visual analysis

of the jugular pulse waves, is of confirmatory value. By the use of graphic methods, the diagnosis is easy and certain.

Premature beats of frequent occurrence at times may simulate closely the arrhythmia of fibrillation. The differential diagnosis has been discussed under premature beats.

**PROGNOSIS.**—Fibrillation is usually permanent, but may be transient. If at all persistent it is reasonable to presume important damage to the myocardium. Much improvement may result from the presence of heart-block produced by drug therapy, or, as sometimes happens, resulting from natural causes. Then the efficiency of the heart depends in large measure upon the integrity of the ventricular muscle. In other words, if the fibrillation is controlled by efficient treatment, the results are often very gratifying, but the latter must not be expected if the ventricular muscle is much impaired.

**TREATMENT.**—The first principle in the treatment of auricular fibrillation is to administer digitalis to the point of inducing sufficient heart-block to prevent some of the shower of impulses, coming down from the auricle, from reaching their destination. By so doing, the ventricle is allowed more rest, and has time to fill properly before contracting. Sometimes heart-block occurs from natural causes, and then the patient is more comfortable, even if taking no medicine.

A case of auricular fibrillation is often one of the most satisfying of heart conditions to treat. The removal of the pulse deficit *in toto*, or in large part, indicates that the correct amount of digitalization is present. The optimum ventricular rate varies from about 60 to 80 per minute. Usually the digitalis should be crowded at first, and, after

digitalization has been achieved, the result may be maintained by a suitable smaller dose. It seems fair to state that the patient is not receiving that to which he is entitled unless the drug is pushed to the point of lowering the ventricular rate, with the removal of all or most of the pulse deficit, and the marked clinical improvement, or until the onset of symptoms or signs of digitalis poisoning.

The treatment of auricular fibrillation by the administration of quinidine sulphate, by which, in successful cases, the normal rhythm is reestablished, is as yet in the experimental stage. The prospect is good of a real advance in cardiac therapy. Quinidine will be further discussed in the chapter on treatment.

In conclusion, it must not be forgotten that the underlying disease of the heart should receive adequate attention.

### HEART-BLOCK

In this condition the essential feature is defective conduction between the auricle and the ventricle.

**ETIOLOGY.**—Heart-block has been produced experimentally by various procedures that interfere with the bundle of fibres conducting between the auricles and ventricles. It may result from stimulation of the vagus nerve. Many poisons may impair the conduction. Prominent among these are: digitalis, strophanthin, aconitine, muscarine, physostigmine, nicotine, potassium salts, morphine, and adrenalin. The action of the latter two is by way of the vagus nerve,<sup>13</sup> while many of the others act directly on the conduction tissues. Heart-block occurs in asphyxia, anaphylaxis, and diphtheria. The most common causes in

<sup>13</sup> LEWIS, THOMAS: The Mechanism and Graphic Registration of the Heart Beat, Paul B. Hoeber, New York, 1920.

clinical cases are syphilis, arteriosclerosis, rheumatic heart disease, and as a result of the administration of digitalis.

**PATHOLOGY.**—The findings are inconstant. Partial to complete destruction of the fibres of the bundle of His may be found. In some cases, the destruction of part of the fibres may so delay the conduction of impulse from the upper part of the heart, that the ventricle may contract in response to stimuli originating in its own tissues below the lesion. Thus it is possible to have complete heart-block in life, although at autopsy the bundle is not completely destroyed. Of course, if the conduction defect is due solely to vagus action, or to the administration of digitalis, the condition cannot be recognized by the pathologist. In disease the lesions are rarely limited to the specialized tissues, but other parts of the heart are also involved, showing the changes of syphilis, rheumatic infection, arteriosclerosis, or whatever affection may be responsible for the heart-block.

**SYMPTOMS AND SIGNS.**—Some degree of heart failure is usually present, though some patients, whose life is not too strenuous, may be remarkably comfortable.

On the other hand, grave symptoms, referable to the nervous system, are not uncommon. In such cases, a marked slowing or standstill of the ventricle produces an insufficiency in the supply of blood to the higher nervous centres, and the symptoms of cerebral anaemia result. These symptoms, depending on the length of the attack, are: pallor, transient giddiness or dimness of vision, momentary loss of consciousness, convulsions, and, in the end, death. According to Lewis,<sup>13</sup> if the standstill of the ventricle lasts but two or three seconds, there is little disturb-

ance. If the pause is for three to five seconds, a momentary loss of consciousness occurs. If the ventricle fails to beat for a longer period, as from fifteen to twenty seconds, twitchings or convulsive movements appear, the breathing deepens and becomes sighing, and cyanosis gradually develops. If the heart beat is in abeyance for ninety to one hundred and twenty seconds recovery is rarely witnessed.

When complete heart-block is present, the pulse and ventricular rhythm are regular and of a rate averaging 30 to 40 per minute. Higher and lower rates occur. During the diastoles, the auricular contractions may sometimes be heard as faint short sounds. Inspection of the jugular pulse may disclose waves during the long pauses. Since the ventricles are then in diastole, these waves must be due to contractions of the auricle: they can only be confused with the "h" or "b" wave, which is a single wave occurring in early diastole, at about the time of the third heart sound. When the auricular systole coincides with that of the ventricle, careful auscultation may note a slight accentuation of the heart sound.

In incomplete heart-block, the pulse may be irregular due to gaps caused by dropped beats. During these pauses, auscultation of the heart discloses a silent period, or, in some instances, the faint auricular sound may be audible. In other cases, there may be no dropped beats and the only suggestion of partial heart-block may be from the separation of the auricular and ventricular sounds, and, if the former is audible, a triple or gallop rhythm may be noted.

As a result of exercise, in heart-block, shortness of

breath tends to appear readily. If the block is complete, the heart rate does not increase.

**DIAGNOSIS.**—A slow, regular rhythm is strongly suggestive of complete heart-block. When the disturbance is limited to some delay in conduction, the condition may not be recognized unless a polygram or electrocardiogram is obtained. Gallop rhythm may indicate partial heart-block. Dropped beats are due to heart-block and, exceptionally, due to sino-auricular block. Their differentiation from premature beats has been discussed under the latter arrhythmia. In practice, the true dropped beat is relatively uncommon. Its confusion with an extrasystole is so frequent, and the difference in prognosis and treatment of the two conditions is so great, that it is suggested that the diagnosis of dropped beat be confirmed by the use of graphic methods. The distinction certainly cannot be made by palpation of the radial pulse.

In electrocardiography, the subcutaneous injection of gm. 0.002 to 0.0015 (gr.  $\frac{1}{30}$  to  $\frac{1}{40}$ ) of atropine sulphate will abolish the vagus influence on the heart and determine, in an electrocardiogram taken about 20 to 30 minutes later, what part, if any, of the heart-block is due to the action of the vagus nerve.

*Adams-Stokes' syndrome*, in which a slow pulse is associated with fainting attacks, is thought to have been due to heart-block and syncope, as described above. However, extreme bradycardia and the attending symptoms are not always due to heart-block. Frequent extrasystoles may retard the pulse rate (though not that of the ventricle), and the accidental association of epilepsy of nervous origin

with such a case has been reported.<sup>14</sup> There are a number of conditions in which Adams-Stokes' syndrome may appear, though there may be no impairment in the auriculo-ventricular conduction.

It should be remembered that an irregularity caused by heart-block is a regular irregularity. The author has met an instance where digital pressure on the vagus in the neck caused a prompt change from a one to one to a two to one rhythm and gave a superficial resemblance to the control of a paroxysm of tachycardia.

**PROGNOSIS.**—This is largely dependent upon the heart affection of which heart-block may be but one feature. Heart-block due to actual lesion of the conduction tissues is usually permanent, if it has already persisted more than perhaps a few weeks; in certain acute conditions, much improvement may occur spontaneously with the convalescence of the affection, usually an infection, of which it is a feature. Heart-block of organic causation is always a serious condition; sudden death is not uncommon, and if the block persists, survival beyond one to three years is the exception. There are, it is true, cases in which the patient lives in comparative comfort for years.

Transient heart-block due to vagus stimulation or to the effect of digitalis is, of course, in a different category. That due to digitalis may be produced intentionally and is often beneficial in controlling an arrhythmia, especially that of auricular fibrillation. Natural heart-block is likewise helpful here.

**TREATMENT.**—Treatment is primarily that of the underlying disease. All cases of heart-block, not clearly

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<sup>14</sup> LEWIS, THOMAS: *The Mechanism and Graphic Registration of the Heart Beat*, Paul B. Hoeber, New York, 1920.

of other causation, and occurring in patients in the age limits of about twenty to sixty years, should be subjected to the therapeutic test for syphilis. The reasons for this have been emphasized under cardiovascular syphilis; perhaps the chief point is that luetic heart disease tends to progress, and the aim should be to prevent further impairment of the integrity of the heart.

Heart-block due to poisons, or the overuse of digitalis, will disappear by cessation of the drug. Atropine sulphate gm. 0.0006 to 0.0005 (gr.  $\frac{1}{100}$  to  $\frac{1}{120}$ ), three to four times daily, will lift the vagus action on the conduction tissues, and remove that factor in the causation of the heart-block. When the symptoms associated with impairment of the blood supply of the brain are present, the recumbent posture is indicated.

It may seem paradoxical to state at one time that heart-block is a serious disturbance and requires treatment, and in the next breath, almost, to advocate the production of heart-block by digitalis therapy. In explanation of this it should be recalled that study of the physiology of exercise discloses that the heart may accomplish the needed increase in the output of blood per minute by a greater output per beat, and by an acceleration of the rate, *i.e.*, more beats per minute. If the presence of heart-block prevents a ready increase in the heart rate, it becomes apparent that a heart so affected will be less able to meet the physiological demands in exercise. Therapeutic heart-block is indicated to combat certain unfavorable features of undue acceleration of the heart rate, as described under fibrillation of the auricles. Heart-block is then the lesser of two evils.

## PULSUS ALTERNANS

Pulsus alternans can hardly be diagnosed save by using instrumental means of examination. It is best demonstrated in the arterial pulse tracing, but occasionally is present in the electrocardiogram. It may also be detected in the taking of the systolic blood pressure by noting that the number of beats coming through under the blood-pressure cuff is suddenly doubled as the pressure falls about five or more millimetres. Certain types of extrasystoles, namely, the interpolated, and those causing a bigeminal pulse, may be confused with true alternation. The use of graphic records will make the differentiation.

The exact mechanism of alternation is hypothetical in part; the condition is generally ascribed to a variation in the contractility of the ventricular muscle. The ventricle, as is shown in the electrocardiogram, contracts regularly, but there is an alternation in the amount of blood expelled into the aorta.

Alternation may be found when an apparently healthy heart is overtaxed, especially when beating at a rapid rate, and also in hearts in which the muscle is seriously impaired. It is particularly common in association with the changes in hypertensive heart disease, and in the arteriosclerotic heart, in which it may be associated with anginoid pain. P. D. White<sup>15</sup> reported its presence in one-third of the cases showing any degree of heart failure, in which he obtained pulse tracings.

The detection of alternation is of importance chiefly from its bearing on prognosis. The majority of patients who exhibit the phenomenon of pulsus alternans succumb

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<sup>15</sup> WHITE, P. D.: *Amer. Jour. Med. Sci.*, cl: 82, 1915.

within a few years. The writer knows of a case in which alternation has been present most of the time for a period exceeding six years.

The treatment is that of the associated heart affection.

### COMBINED ARRHYTHMIAS

In the above description, the various functional conditions of the heart have been described as occurring singly, but in practice it is common to find two or more affecting an individual patient. The relationship between premature beats, paroxysmal tachycardia, auricular flutter, and auricular fibrillation, is so close that a single patient may be affected with all of these four arrhythmias at various times. The dependence of paroxysmal tachycardia on premature beats has been mentioned above. The fact that circus motion is concerned in the true mechanism of auricular flutter, and, with the addition of conduction defects, in that of fibrillation of the auricles, has been pointed out. Alternation of the pulse is often more marked following extrasystoles, or a paroxysm of tachycardia; in fact, alternation may occur in the waves during a paroxysm. Most any of the arrhythmias may alternate with the normal rhythm from the sino-auricular node.

Some degree of heart-block is the rule when the heart is dominated by flutter of the auricles, and the same conduction defect is common in cases of auricular fibrillation. In the latter arrhythmia, when the ventricular rate is kept down by heart-block of natural or digitalis causation, extrasystoles may also appear. A well-known example of this is the bigeminy or coupling effect following the free use of digitalis in the treatment of fibrillation. A moment's

reflection will make it clear, as is demonstrated by the electrocardiogram, that since the auricles are in the grip of fibrillation, the premature beats must be of ventricular origin.

Further examples could be given, but space forbids. The interpretation of combined arrhythmias is, needless to say, often difficult. The elucidation of some of these complex problems in cardiac diagnosis is one of the most interesting and instructive of the contributions of the graphic methods to clinical medicine.



**SECTION IV**  
**STRUCTURAL LESIONS**



## CHAPTER XVIII

### STRUCTURAL LESIONS

IT WOULD seem that until recently there has been a tendency to overemphasize the structural pathology of the heart. As urged in the chapter on "The Classification of Heart Disease," it is better to think primarily in terms of the type of heart disease, which expresses the etiology when known, and then of the functional condition and structural changes. Realizing, however, the limitations to the above idealistic conception of heart affections, the writer will attempt in this section to present briefly data, pertaining to the structural pathology, which may be of value in diagnosis, or which may not have been sufficiently discussed in the preceding chapters. Although, as emphasized by James Mackenzie, disease processes are rarely ever limited to a single layer of the heart tissues, for the purpose of convenience, the changes in the individual layers of the heart will be presented separately.

#### ENDOCARDIAL LESIONS

ACUTE ENDOCARDITIS, as a clinical problem, is largely a matter of acute rheumatic heart disease, or acute septic heart disease. One or more murmurs are usually audible on auscultation, but it is often a matter of judgment as to whether they signify actual changes in the valvular endocardium. Their persistence or disappearance after a sufficient period of time (after convalescence is well established), and the development or absence of other signs and symptoms, are the safest evidence on which to make

the decision. Acute endocarditis has been sufficiently described under the above infections of the heart and need not further detain us here.

**CHRONIC ENDOCARDITIS** is another term for chronic valvular heart disease, and may be considered under the lesions of the respective valves. The symptoms of chronic valvular disease show little that is distinctive of the particular valves involved. It is usually by the physical signs that they are distinguished.

**AORTIC REGURGITATION.**—Insufficiency of the aortic valve in adults is usually syphilitic, whereas the rheumatic infections predominate as the cause in children. When present in rheumatic heart disease, it is often associated with mitral lesions. Insufficiency and stenosis are not nearly so frequently associated in the aortic valve as are these two conditions in the mitral valve. This seems explicable in part from the fact that aortic insufficiency is usually syphilitic, and this disease (see pathology of cardiovascular syphilis) rarely produces stenotic change in the orifice.

The best sign of aortic insufficiency is a diastolic murmur beginning with the second sound, and of maximum audibility along the left sternal margin, at the third and fourth costal cartilages. The murmur is usually audible also at the second right costal cartilage, and at times along the left border of the heart and apex, where it is to be distinguished from the diastolic of mitral origin by its earlier commencement, higher pitch, and more blowing quality. As noted in the chapter on "Methods of Examination," the murmur of aortic regurgitation, in some cases, is detected only in case the examiner applies his ear direct to the chest, or uses the diaphragm end piece (Bowles) of

the stethoscope with the patient in the erect or leaning-forward posture. The aortic second sound is usually weakened or absent, but may be of normal intensity.

Cardiac enlargement develops sooner or later, if the regurgitation is extensive; it is in just such cases that *cor bovinum* (ox heart), the largest type of heart, is found. The apex impulse may be displaced downward to the sixth or seventh intercostal space, and to the left close to the anterior axillary line. To the palpating hand, the impulse feels forceful and sustained. On Röntgen examination, the cardiac outline shows enlargement downward and to the left, with a somewhat blunted apex, and the shadow of the aorta is broadened, with increased pulsation, when examined fluoroscopically.

A slight amount of regurgitation through the aortic valve, however, may exist without any obvious enlargement of the heart.

Various VASCULAR PHENOMENA are noteworthy features in patients in whom the aortic valve is insufficient. They are valuable confirmatory evidence of incompetency of the aortic valve, but are not pathognomonic unless well marked, as a Corrigan pulse, and even visible capillary pulsation may be present in individuals with low vasomotor tone, in some cases of exophthalmic goitre, in neurasthenia, in congenital heart disease with a patent ductus arteriosus,<sup>1</sup> etc.

The water-hammer, or *Corrigan pulse*, is one in which there is a quick rise and fall, so that it is often described as a collapsing pulse. Its chief feature, as shown by blood-pressure studies, is a lowering of the diastolic level,

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<sup>1</sup> WALKER, R. W. S.: *Heart*, ii, 3: 249, 1911.

while the systolic remains essentially unchanged. When present, a visible throbbing of the carotids and other superficial arteries may be observed. A slight nodding or shaking of the head accompanies well-marked cases.

The capillary pulse may be seen: (1), in the systolic flushing of a line drawn across the forehead by the finger or by a blunt instrument; (2), if the end of the patient's fingernail is bent to the point of just blanching the pulp beneath; (3), as a faint pulsation, if a glass slide be pressed against the mucocutaneous margin of the lower lip of the mouth. The last method is usually the best.

The systolic pressure in the femoral artery is often considerably in excess of that in the brachial (sometimes 100 mm. of mercury higher). In explanation of this Starling<sup>2</sup> points out that the femoral artery is more rigid than the brachial, and the peripheral resistance is more definitely localized, so that the reflected waves in the artery occur in so short a time after the primary wave has passed down, that there is a summation of the two waves, with the production of a higher maximum and lower minimum pressure than was present in the waves as started in the aorta.

On auscultation of the peripheral arteries, further phenomena of interest may be noted. This is usually most successfully performed if the bell of the stethoscope is placed over the femoral artery in Scarpa's triangle.

A sharp sound, called the *pistol-shot sound*, may be detected when the end of the stethoscope is in position over the artery, and this gives way to a systolic murmur if pressure is made by the instrument on the underlying

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<sup>2</sup> STARLING, E. H.: Human Physiology, 3rd edition, 1920, p. 971.

vessel. As the pressure is continued, a diastolic murmur may appear in addition, thus giving a to-and-fro murmur. This diastolic murmur, termed *Duroziez's sign*, occurs only in cases showing a marked Corrigan pulse, with a low diastolic pressure level, and is of greater value than is the pistol shot as an indication of insufficiency of the aortic valve. Duroziez's sign is often confused with *Traube's sign*, which is a double sound, not murmur, heard when the stethoscope is placed, but not pressed, over the artery. It is a rare sign and present only in extreme degrees of the water-hammer pulse.

Functional insufficiency of the aortic valve is rare, but has been confirmed at autopsy. It has also been produced experimentally.<sup>3, 4</sup>

A word should be said about the *mechanism of the vascular changes* in aortic insufficiency. Since the time of Corrigan, it has been generally accepted that the water-hammer pulse is due to the actual backward flowing of the blood, but, as a result of experimental work, Stewart<sup>4</sup> concludes that this type of pulse is caused by a vasomotor reflex which dilates the peripheral vessels. Stewart and Zollinger<sup>5</sup> have proven that the amount of blood which actually regurgitates into the ventricle measures but a few cubic centimetres. The left ventricle, then, still receives the bulk of its content of blood in the usual way, *i.e.*, through the much larger aperture, the mitral valve. Wiggers<sup>6</sup> admits that there is no essential backward flow

<sup>3</sup> THAYER, W. S., and MACCALLUM: *Amer. Jour. Med. Sci.*, cxxxiii: 249, 1907.

<sup>4</sup> STEWART, H. A.: *Arch. Int. Med.*, 1: 102, 1908.

<sup>5</sup> ZOLLINGER, F.: *Arch. f. Exper. Path.*, 61: 193, 1909.

<sup>6</sup> WIGGERS, C. J.: *Modern Aspects of the Circulation in Health and Disease*, Lea & Febiger, 1913, p. 324.

of blood and suggests that such would be physically impossible in the time allowed, since the velocity of the blood flow is only about three-tenths of a metre per second. The pulse wave, however, according to Wiggers, travels at a rate of six to ten metres per second, and, if the writer reads this physiologist correctly, the latter explains the Corrigan pulse as directly due to a retrograde pressure wave. Stewart also believes that the arterial pressure is exerted backward into the cavity of the left ventricle, but holds that this retrograde pressure excites the vasomotor reflex, which in turn produces the Corrigan pulse. His paper contains considerable data in support of this contention. That the cardiac output is not materially increased is a further observation in the experiments of Stewart and Zollinger.

**AORTIC STENOSIS.**—Stenosis of the aortic valve is much less common than is aortic insufficiency, the latter occurring about ten times as frequently. Aortic stenosis is often diagnosed when it does not exist; aneurism, aortic dilatation, the anaemias, etc., may also cause a systolic murmur at the second right costal cartilage. Pulmonary stenosis, with its systolic murmur and thrill, may be differentiated by noting that these findings have their point of maximum intensity to the left of the sternum and the murmur is not transmitted to the neck. At best the diagnosis of aortic stenosis is often difficult or uncertain, but this is less unfortunate, since this lesion is almost always a manifestation of rheumatic heart disease, and other evidence of that affection is generally present. Syphilis almost never causes obstruction of the aortic orifice.

A rough systolic murmur, accompanied by a systolic

thrill, over the aortic area and transmitted to the vessels of the neck, usually indicates aortic stenosis. If the pulse is anacrotic and plateau in type, the diagnosis obtains strong support, but as insufficiency of the valve usually complicates, the characteristic pulse findings may not be present. The bisferiens pulse, in which the primary wave in the arteriogram has a bifurcated summit, has been attributed to aortic stenosis, but its value in the diagnosis of this valve change apparently is doubtful. The second sound is often weak or absent, but distinct exceptions occur. The heart is usually enlarged downward and to the left, but never to the advanced degree found in some cases of aortic regurgitation. The electrocardiogram usually shows left ventricular preponderance. There is nothing characteristic about the blood pressure. On Röntgen examination, the first part of the aorta does not show enlargement to the extent so often present in aortic insufficiency.

**MITRAL REGURGITATION**, due to actual change in the valve curtains, is probably always due to rheumatic heart disease, or its more severe type, termed septic heart disease in this book. Insufficiency of this valve usually occurs in conjunction with some degree of stenosis, but the latter is often not diagnosed, due to the fact that sufficient time (a few months) has not elapsed for it to develop, or, in other cases, when present, it is not easily detected. For further data on organic (in the sense of actual valve change) mitral insufficiency, the reader is referred to the chapter on rheumatic heart disease.

Mitral regurgitation due to imperfect muscular co-operation in the closure of the valve is common. It occurs

in most febrile diseases, at times in all of the types of heart disease previously described, and there is good evidence that a small degree of it may be present even in some hearts which are properly classified as normal. It may be diagnosed from the presence of a systolic murmur at the apex, not accompanied by evidence of enlargement of the heart. In some types of heart disease, however, as in cardiovascular syphilis and hypertensive heart disease, cardiac hypertrophy may be present, and yet an apical systolic murmur does not signify an actual valve lesion. A history of an infection of the rheumatic type should imply caution in assessing a systolic bruit as not due to valve damage. If a febrile disease is present it is best to withhold decision until it is evident that an infection has not attacked the valve.

An error in the differential diagnosis between mitral insufficiency of non-valvular causation, and that due to actual lesion of the valve flaps, providing the process attacking the valve is at an end, is not of serious importance; for if the damage that an infection has done is limited to the production of mitral insufficiency, the integrity of the heart is but little impaired.

**MITRAL STENOSIS** has been sufficiently discussed under rheumatic heart disease.

**TRICUSPID LESIONS** are relatively rare, and when their clinical diagnosis is submitted to confirmation by post-mortem examination of the heart, the results are often disappointing. Structural change in the cusps of the tricuspid valve is almost always due to rheumatic or septic heart disease. It rarely occurs alone, but is associated with lesions of the mitral valve, sometimes of both the mitral and aortic valves, and less frequently of the pulmonic valve in

addition. The murmurs are soft, faint, blowing, rarely rough, and usually more superficial and shorter than the synchronous mitral murmurs. The relatively lower pressure which obtains in the right side of the heart is probably the chief factor in influencing the quality of the murmurs.

**TRICUSPID STENOSIS**, like mitral stenosis, produces a mid-diastolic murmur, with or without a presystolic phase, and localized over and just to the left of the lower half of the sternum. It must be differentiated from a widely transmitted diastolic of mitral stenosis, and from that of aortic insufficiency, which latter murmur is audible both in the same place and higher up on the sternum, and, what is more important, always begins earlier in diastole.

**TRICUSPID REGURGITATION**, due to organic change in the valve, usually accompanies the stenotic change, and much that has been said about the latter is likewise applicable. Its murmur is, of course, systolic and located over the tricuspid area.

There are other signs of organic lesions of the tricuspid valve. Enlargement of the liver is often present, but exceptions occur. Transverse enlargement of the heart, increased dulness to percussion to the right of the sternum (over the right auricle), and a prominence of the Röntgen shadow of the right auricle, occur. The liver may be increased in size and pulsate. If there is a prominent wave due to auricular contraction, Mackenzie holds it as possible evidence of organic tricuspid stenosis. Dilated cervical veins, cyanosis associated in some cases with a slight jaundice, polycythemia, and clubbing of the fingers, are found in some of the more advanced cases.

Tricuspid regurgitation, in cases in which the valve curtains are normal but the valve incompetent because

of failure of the muscular coöperation in the closure of the valve, is, as in the case of the mitral valve, much more common. It probably occurs, to some degree, in a large proportion of the cases of advanced heart failure, and perhaps in some others. The diagnosis, as made clinically, is usually based on non-cardiac data, for example, symptoms of heart failure associated with prominent pulsation of the cervical veins, enlarged and pulsating liver, and sometimes ascites. If, as is often true in these cases, auricular fibrillation is present, the venous pulse will be of the ventricular type, and the symptoms of heart failure, as urged by Mackenzie,<sup>7</sup> are due to the fibrillation. The heart failure is never due solely to the tricuspid regurgitation. It is the failure of the heart to propel the blood forward, and not the leakage backward of some blood, that counts. If the auricle is acting, there is no ventricular jugular pulse and then the tricuspid regurgitation is of little importance.

**PULMONARY VALVE LESIONS.**—Endocarditis of the pulmonary valve is very rare. When present it is usually associated with lesions of the other cardiac valves in rheumatic heart disease.

**PULMONARY INSUFFICIENCY** gives the same signs as the Graham Steell murmur described under rheumatic heart disease.

**PULMONARY STENOSIS** as a result of disease after birth, is very rare. It is to be distinguished from the congenital type by the history of the case and evidence of other valvular lesions.

A systolic murmur at the second left interspace is not uncommon. It has been ascribed to dilatation of the conus

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<sup>7</sup> MACKENZIE, JAMES: *Oxford Medicine*, ii: 465, 1920.

arteriosus. Such a murmur may occur in fever and in anaemia. In healthy adults, this systolic murmur may be present during expiration and absent in inspiration. Without other evidence, it in no sense indicates disease, but is probably due to certain conditions in the relation of the conus arteriosus and pulmonary artery to the overlying chest wall. For further data on pulmonary stenosis, and systolic murmurs localized at the pulmonic area, the reader is referred to the chapter on congenital heart disease.

### MYOCARDIAL LESIONS

MYOCARDITIS, as a clinical diagnosis, has been much abused. In some instances it would seem reasonable to suppose that the user's mind worked somewhat as follows: "I think there is something wrong with the heart. I can't hear any murmurs so I can't blame it on a valve lesion; I don't know what the trouble is, so I guess I'll just say it is myocarditis." Thus the term myocarditis has been applied to cases of cardiovascular syphilis, arteriosclerosis, hypertensive heart disease, etc., and even to cases in which the heart was normal. In this sense myocarditis has, at times, been used as a wastebasket diagnosis.

Myocarditis is defined in the dictionary as inflammation of the myocardium. Of course it occurs, and in fact it forms an important part of the pathologists' descriptions of diseased hearts. As several times previously stated, Mackenzie has emphasized the involvement of the myocardium in practically every case in which the valvular endocardium was attacked. The writer wishes to do nothing to lessen Mackenzie's well-placed emphasis, but believes it will make for clearer thinking in heart affections if the term myocarditis is used circumspectly, as part of a larger

diagnosis, perhaps, and only after the case has been carefully studied. In most instances, such study will be rewarded with the finding of evidence sufficient to base a diagnosis of one of the conditions described in the section on functional conditions, or of a definite type of heart disease, or, less frequently, of no heart disease. In the preceding chapters numerous references will be found to the various lesions of the myocardium.

**HYPERTROPHY.**—Much that has been written on the subject of cardiac hypertrophy is based upon the mechanical conception of changes produced by valvular lesions, etc., but to-day this has been largely abandoned in favor of that of infection. Great enlargement of the heart may be present in cases in which the valve lesion is insignificant. There is an unknown factor in the causation of hypertrophy.

Hypertrophy and dilatation are often present in the same heart, and it is rarely possible to determine accurately the extent of either. Most of the physical signs serve to indicate cardiac enlargement, but not the relative part of hypertrophy or of dilatation. It is well known that dilatation is a feature of the normal physiology of exercise.

The importance of the subject is such that it may be well to present some of the observations made by Lewis,<sup>8</sup> and supplemented by Cotton,<sup>9</sup> on hypertrophy of the heart. Lewis pointed out that when the heart is inspected in the fresh state at autopsy, and even when the organ has been cut in several planes, the degree of hypertrophy of the muscle and its relative excess in one or the other chamber are but imperfectly determined. These workers,

<sup>8</sup> LEWIS, T.: "Observations upon Ventricular Hypertrophy, with Especial Reference to Predominance of One or Other Chamber," *Heart*, v: 367, July, 1914.

<sup>9</sup> COTTON, T. F.: *Heart*, vi: 217, Jan., 1917.

therefore, separated by a special dissection the musculature of each ventricle and weighed them separately.

According to Lewis, the commonest type of hypertrophy is that in which the heart is uniformly hypertrophied. In renal disease it is the rule. And in lesions of the aortic valve, uniform hypertrophy is almost as frequent as that in which it is more marked in the left ventricle. In aortic cases, Cotton found instances in which the right ventricle was hypertrophied in excess of that in the left side of the heart.

It was further shown that a visible pulsation or palpable thrust in the epigastrium is not a particularly valuable sign of hypertrophy of the right side of the heart. A forcible and thrusting maximum impulse, displaced downward and to the left, is a somewhat more reliable sign of left ventricular hypertrophy.

The evidence of ventricular preponderance in the electrocardiogram follows the ventricular weights quite accurately.<sup>10</sup> However, in uniform hypertrophy, the electrocardiogram shows no preponderance.

## PERICARDIAL LESIONS

Acute, and chronic or adhesive pericarditis, have been discussed in part under rheumatic heart disease.

**ACUTE PERICARDITIS.**—It is generally a condition secondary to some other disease and consequently often escapes diagnosis. Thus it is more often disclosed at autopsy than diagnosed clinically. In the majority of instances, pericarditis is due to the rheumatic type of infection, but rarer forms occur. Thus, in pulmonary tubercu-

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<sup>10</sup> More recently exception has been found to this. Wilson and Hermann, see reference nine under Ventricular Preponderance, chapter iv.

losis, the pericardium may show a few tubercles, or be entirely replaced by a thick layer of fibrous and caseous material. Carcinoma may invade the pericardium. An occasional cause is infection following a wound, such as a stab wound. A dry pericarditis may occur as a late complication of nephritis.

Williamson<sup>11</sup> has shown experimentally that the pericardial sac can hold 100 c.c. without being stretched, but when 655 c.c. was exceeded, the pericardium ruptured or was torn off the great vessels. The fluid accumulates at first near the apex and below, and usually pushes the left lobe of the liver downward. Later, as the amount of the fluid increases, it accumulates over the great vessels. *Rotch's sign* of obliteration of the cardiohepatic angle, as determined by percussion, is present only in the large effusions, and is not found in the Röntgen examination.

Large effusions compress the adjacent lung so that at times there is an area below the left scapula over which dulness, increased fremitus, and bronchial breathing, closely simulate a pneumonic process. This is usually designated as *Ewart's* or *Bamberger's sign*. The findings may lessen if the patient can assume the knee-chest posture. At times the doubt as to the presence of a pleuritic effusion is only settled as a result of a dry tap or exploratory thoracentesis.

The pericardial friction murmur or rub varies from a feeble grazing sound to a loud grating or creaking which is sometimes palpable to the hand. It is not in definite accord with the heart sound and is liable to vary much in intensity from moment to moment. On expiration or firm pressure with the stethoscope, the murmur is commonly

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<sup>11</sup> WILLIAMSON, C. S.: *Arch. Int. Med.*, 25, 2: 206, 1920.

intensified. Perhaps the point of greatest assistance in differentiating a pericardial friction sound from an endocardial murmur is that the former has a distinctly superficial quality; it sounds as though near the ear. If a rub is heard only near the cardiac border it may not indicate pericarditis, but may be of pleural origin, *i.e.*, the pleuro-pericardial friction sound.

If the heart is large, its anterior surface is generally in contact with the chest wall, and so a pericardial rub may be audible even in the presence of an effusion of considerable size.

Percussion dulness extends well outside the apex impulse, and in large effusions the latter may become obliterated and the heart sounds feeble. Dilatation of the heart and pericarditis with effusion, at times are differentiated with difficulty. This uncertainty may exist even on radiographic examination, unless the plate clearly shows the outline of the heart separated by a lighter area from the outline of the pericardium.

Treatment is, of course, based on the underlying disease. If acute, rest in bed, usually with a headrest, is essential. Pain should be met by an icebag to the precordia and sufficient sedatives, usually morphin or codein.

The effusion of rheumatic origin will usually subside spontaneously, but if large, and there are signs of undue cardiac embarrassment, resort should be made to tapping of the pericardial sac. The best points at which to insert the aspirating needle are in the fifth left (or right) intercostal space, about an inch from the sternum. Paracentesis at either of these points will avoid injuring the internal mammary arteries, which lie about one half inch from the sternum, and will not pass through the pleural

cavity. About equally good is Sears' point, outside the nipple or midclavicular line, and inside the limit of cardiac dulness. Under local anaesthesia and aseptic precautions, the needle should be passed with care and gentleness, to enable one to feel the giving way of the parietal pericardium, and to avoid cardiac trauma. Removal of but a small quantity frequently provokes absorption of the effusion. If the fluid reforms with recurrence of symptoms, a repetition of the paracentesis may become necessary.

Purulent pericarditis requires surgical drainage of the pericardium. If done promptly and well, the prognosis is by no means hopeless.

**CHRONIC ADHESIVE PERICARDITIS** has been sufficiently described in an earlier chapter (see rheumatic heart disease). A few cases in which symptoms were marked have been treated surgically. The adhesions tend to reform, and unless the ribs overlying the heart are removed, the results are problematical. Opinions differ as to the value of this operation. The heart muscle must, furthermore, be in good condition to warrant surgical interference.

**PNEUMOPERICARDIUM**, or hydro- (or pyo-) pneumopericarditis, is a rare condition. It may result from gas formation occurring after death. In life it may be produced by perforation of a viscus, abscess, pulmonary cavity, knife in the esophagus, etc. A large accumulation of gas in the vicinity of the heart may simulate the above rare pneumopericarditis. The treatment should be surgical if the condition of the patient warrants interference.

**HEMOPERICARDIUM**.—The exudate is tinged with blood in certain forms of pericarditis. A large quantity of blood may escape into the sac in wounds, rupture of the heart, or rupture of an aneurism. The prognosis is favorable only

in some of the traumatic cases, an increasing number of which have been relieved by surgery undertaken to repair the often associated wound of the heart. In rupture of the heart or of an aneurism into the pericardium, death results promptly or may be delayed a few hours to possibly a few days.

### ANEURISM

Aneurism is usually a manifestation of syphilis, and it has accordingly received some attention in the chapter on cardiovascular syphilis.

An aneurism may be defined in simple terms as a sac formed by the dilatation of the walls of an artery, and filled with blood. Many terms have been employed to describe aneurisms according as they differ in shape, size, etc., but perhaps the most useful are: sacculated, fusiform, dissecting, true, and false. A *sacculated aneurism* is one in which the sac-like dilatation opens into the artery by an opening which is small compared with the size of the sac, while the *fusiform type* is merely a spindle-shaped dilatation of the artery. The *dissecting aneurism* is one in which the blood forces a passage between the coats of the arterial wall; in some instances a second opening may exist by which the blood may reenter the lumen of the affected artery. An aneurism is described as *true* when the wall of the sac is formed by one or more of the walls of the artery; while in contrast to this the aneurism is said to be *false* in case the walls of the vessel are broken through and the blood retained by the surrounding tissues. From the above, then, it should be clear that there is much variation in the shape, size, and nature of the wall surrounding the different aneurisms.

The statement that an aneurism is usually the result of invasion of the arterial walls by the pale spirochete is particularly true of the aorta above the level of the diaphragm. Its rare occurrence in other infections has been mentioned (see cardiovascular syphilis). Whether arteriosclerosis ever causes an aneurismal change, is, in the light of modern pathology, problematical. Before accepting a given aneurism as due to arteriosclerosis, the tissues should be studied histologically to exclude syphilis.

Aneurisms occurring in the peripheral arteries are beyond the limits set in this book.

Many aneurisms are diagnosed in life only as a result of Röntgen examination. Even then, one involving the descending thoracic aorta has escaped detection until the patient was examined in an oblique position. Symptoms may arise only as a result of pressure on adjacent structures, and vary according to the nature of the latter, and the rapidity of growth of the aneurism. In those aneurisms which approach the surface, and may be examined by palpation, the demonstration of an impulse that is clearly expansile may clinch the diagnosis. This may be demonstrated by placing a finger of each hand on the swelling and noting if they are separated with each pulsation. The same may be accomplished by suitably placing marks with a skin crayon and observing if they rhythmically separate and approach. A valuable sign of aneurism of the transverse arch of the aorta is the tracheal tug. It is best detected by lifting the cricoid cartilage upward between the thumb and forefinger, and noting if there is a downward movement of the larynx with systole. The erect posture is often essential and the head should not be thrown

backward. The phenomenon is due to the pushing down of the left bronchus by the pulsation of the aneurism.

Rupture of the aneurism, with fatal hemorrhage, occurs in about 75 per cent. of the cases. Favorite locations for the hemorrhage are: into the pleural cavities, the pericardium, the left lung, through the skin, and into the esophagus or trachea. Death may also result from suffocation, cachexia, or as a result of emboli.

**SURGICAL TREATMENT**, in a limited number of cases, has resulted in relief of pain and prolongation of life. The measures resorted to are reinforcement of the aortic wall by wrapping a strip of fibrous tissue around it, and resection of the sympathetic nerves close to the aneurism, or of the sympathetic trunk in the neck. Treatment by the introduction of gold-platinum wire and electrolysis is a method which has benefited some cases. Its purpose is to promote coagulation of the blood, and the obliteration of the sac, by organization of the thrombus. It is most likely to succeed in cases in which the aneurism is of a definite sacculated type. The above surgical measures, with the exception of the operation of dividing the cervical sympathetic, are dangerous procedures, and perhaps their greatest justification is in cases attended with severe pain.

**MEDICAL TREATMENT** is usually symptomatic. The iodides in 0.6 to 1.3 gm. (10 to 20 gr.) doses appear to alleviate the pain. Treatment by rest, low diet, and limited fluid, over a period of months, has been advocated, in order to encourage healing by thrombosis. Such treatment has a chance of success only in cases of sacculated aneurism with a small neck. Active individuals will not tolerate this method of treatment.

*Cardiac aneurism* is a rare condition. It is most commonly located in the wall of the left ventricle, near the apex. An infarct of the myocardium, or syphilis, causes most of the cases. When the aneurism is located in the interventricular septum, the conduction tissues are involved. Rupture into the pericardial sac may terminate those involving the external wall of the heart. Needless to say, the diagnosis is rarely made in life.



Courtesy of Frank B. Mallory

PLATE 4. Aneurism of the wall of the left ventricle with thrombus inside, associated with coronary sclerosis and hypertensive heart disease.



## **SECTION V**



## CHAPTER XIX

### TREATMENT

IT IS the purpose of this chapter to present some of the therapeutic measures which, to avoid repetition, have been largely omitted in the earlier part of the text.

The most important principle in cardiac therapy is that the latter should be preceded by the most accurate and complete diagnosis of the status of the heart in question. Adherence to this rule will greatly clarify both prognosis and treatment.

For purposes of convenience, the treatment of heart affections may be roughly grouped under a number of general headings.

#### ETIOLOGICAL TREATMENT

To make this the more obvious is perhaps the strongest argument in support of the method of presentation in this book of diseases of the heart. The section on "Types of Heart Disease," it will be noted, is based on etiology.

*Cardiovascular syphilis* requires treatment directed against the pale spirochete. Suitable measures have already been described and need not be repeated here.

REMOVAL OF A FOCUS OF INFECTION is extremely important in rheumatic heart disease. The most frequent location of such foci is in the tonsils or teeth, but at times it seems fair to incriminate some other locality, such as: the accessory sinuses of the nose, the female pelvis, the prostate, septic wounds, the intestinal tract, etc. The evidence seems strong that the eradication of a focus of

infection lessens the probability of a renewal or increase in the infection of the heart; it is difficult not to believe, in addition, that the prompt treatment of such foci has a prophylactic value in cases in which involvement of the heart may not yet be manifest. In occasional patients, unfortunately, interference with a focus of infection may be followed by an increase in the disturbance of the heart. This possibility may be considered in the prognosis of tonsillectomy, or other procedure, but it should not be allowed to prevent attempts to eradicate foci of infection. Further remarks on this aspect of treatment will be found under rheumatic heart disease.

Infections, however mild, should be treated with greater care when they occur in those affected by cardiac disease. It would seem that if the patient is put to bed, there is less danger that the infection will attack the heart.

### GENERAL TREATMENT

**REST.**—Rest in bed should be the rule in all cases in which fever or sufficient other evidence indicates the presence of an active infection in the heart. In cases presenting the signs and symptoms of grave heart failure, the rest should be as absolute as possible. This means the use of the bedpan, of course, and at times assistance even in shifting position in bed. Decision as to when the patient may properly be allowed up should be based upon the duration and severity of the acute attack, and upon the evidence of cardiac impairment. The bed treatment should continue one or two weeks after cessation of the fever, and in some instances for a distinctly longer period. The process of getting up should be checked by noting the effect on the heart rate, the presence or absence of cardiac

embarrassment, etc., and the amount of liberty should be increased in accordance with the tolerance of the patient to activity.

Rest in bed, however, is by no means of value to each and every case of heart disease. Except for some purpose, such as special study, it certainly is an error of judgment, or an abuse of the patient's trust, to order him to bed solely because of the presence of abnormal physical signs, detected in examination of the heart. The carrying out of treatment by rest in bed imposes so much interference with the patient's affairs, that it should rarely be prescribed, save in the presence of infection, or definite cardiac failure; it should be incumbent on the one who advises bed treatment to show benefit therefrom. Otherwise, in addition to the loss of time, the result may be a loss of general muscular tone, and a lack of confidence of ability to attend to one's daily work.

EXERCISE is of late assuming a more important place in the treatment of heart affections. Together with the gain in the tone of the general musculature of the body, there is often noted an increase in the power of the heart to do its work. This is explainable, in part at least, by the augmentation in the blood supply of the myocardium that is part of the physiology of muscular exertion; if the cardiac tissue has been impaired by inflammatory, or other changes, it would seem helpful to utilize any measures which might insure a better flow of blood through its nutrient vessels. In addition, it is now realized that dilatation of the heart may be a purely physiological process, and thus the fear of this feature has been much lessened.

In practice, the amount of exercise is to be kept well

within the tolerance of the individual patient. The latter may be encouraged to pursue whatever amount of exercise is pleasurable; the symptoms when this limit is exceeded are usually so disagreeable, that the patient will be constrained to cease exerting himself. All forms of competitive exercise, and those in which there is an undue incentive to continue, should be forbidden. The exercise should be repeated daily, and preferably in the open air. Sudden, violent, or excessive forms of physical exertion are seldom wise. Thus, of the games, football and tennis are undesirable, while golf is often excellent. Exercise, then, has become a valuable therapeutic measure; it is often impressive to note how much more efficient the circulation may become, following systematic physical exertion of a suitable type.

**MASSAGE AND PASSIVE MOTIONS.**—These have a moderate use in early convalescence from serious heart failure. As soon as the patient's condition warrants it, active exercise, which is much superior, is permitted. Of course, in patients confined to bed over long periods, massage may do considerable to increase the comfort and to preserve muscular tone. The direction of the massage stroke on the limbs should be towards the trunk, in order to favor the venous and lymph return.

**PSYCHIC ASPECTS.**—As in the treatment of disease in general, attention should be directed towards the mental or psychic attitude of the patient. In cardiology there is the bugbear of the name "heart disease." This term may well be avoided, or, if it creeps in, the physician should take time to make clear the wide range of heart affections, and the lack of wisdom in being influenced by knowledge

of other patients who have been said to be afflicted with "heart disease."

The analogy of valvular heart disease to the "leaky valve" of a mechanical pump has been overused. The term "leaky valve" will be found not infrequently to be associated by the patient with stories of unfavorable outcome in other patients. This expression may be replaced to advantage, perhaps, by that of "scar." Attention can be drawn to the quality of the muscle of the heart, and what work it is able to do. In most instances, also, the physician may, if the subject comes under discussion, honestly point out the unlikelihood of sudden death. A cheerful, hopeful attitude of mind on the part of the patient is just as desirable in cardiac as in other affections.

A considerable part of the treatment in an ambulatory case requires the coöperation of the patient, and to obtain this it is nearly always necessary to give the patient some knowledge of his problem. This should be stated in simple language that is readily understood. Experience will show how much it is profitable to tell the patient; it is rarely wise to dwell upon the unfavorable aspects of heart affections.

The environment of the patient should be considered. Thus, the physician should see that a suitable member of the family has some inkling of the patient's condition, so that the avoidance of certain activities will be attributed to their proper cause, and not to laziness. Effort directed towards the detection of and removal, as far as possible, of unfavorable influences at home or at work, such as is so well done by the social service department in hospital cases, will pay large dividends.

**DIET.**—The principles of this are simplicity, variety, and moderation, especially the latter. Obese patients should be dieted to reduce weight, which is a handicap to the circulation. The undernourished patient may be assisted to become of normal weight; at least it should be ascertained that the patient is on a diet sufficient to maintain good nutrition of the body and likewise of the heart. In bed patients, with marked shortness of breath, food that requires much chewing should be avoided. When edema is present, the daily intake of fluid may be reduced to 800 to 1000 c.c., and salt may be excluded. Dietetic measures are of importance in the treatment of hypertensive heart disease (which see).

**BOWELS.**—Straining at stool is undesirable in cases affected by grave heart failure, and is dangerous if angina pectoris is present. Suitable measures, which do not need description here, should be adopted to prevent the foregoing.

**BATHS.**—Prolonged hot baths are unwise; a tepid, or medium hot, bath is better. Cold baths or showers are suitable only for patients in relatively good condition, and who are accustomed to them. Cold to the skin is contraindicated in angina pectoris.

Baths in which the water contains certain salts, or which is charged with carbonic acid, have been recommended and extensively used at certain health resorts, as Nauheim, etc. There are many other features, such as the freedom from business cares, hygienic mode of life, etc., which doubtless contribute to the benefit that many patients receive. Some of the strongest evidence that the advocates of these baths have to offer is based upon change in the percussion find-

ings, which, the writer would suggest, have distinct limitations as proofs of scientific value. Except at these health resorts, balneotherapy in heart disease has not found much favor.

DRESS should be suitable to avoid chilling. The limbs may require more protection than in normal individuals. On the other hand, the patient should not be burdened with unnecessary weight from the clothing.

CLIMATE.—The patient does not, as a rule, change his residence for climatic reasons. Patients who are much debilitated, and those affected with arteriosclerotic heart disease, or angina pectoris, do better in a moderately warm climate. If such patients live in a region where the winters are severe, they may remain indoors too much, because of the cold; a climate which encourages outdoor life is preferable.

ALTITUDE.—In some normal men, the heart rate has first been found to accelerate when an altitude of five thousand feet<sup>1</sup> was reached. It is well known that those in whom some cardiac defect is present are more sensitive to a change in altitude, but anything up to one thousand feet is usually well tolerated. At a moderate elevation, the more bracing air may have a beneficial effect, and the patient need not, therefore, be restricted to the sea level.

## SYMPTOMATIC TREATMENT

PAIN AND RESTLESSNESS.—When these symptoms are present in bed-patients with grave heart failure, they should be relieved by the administration of an opiate, such as morphin or codein in sufficient amount. The pain that

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<sup>1</sup> SCHNEIDER, E. C.: "Medical Studies in Aviation," *Amer. Med. Assn.*, Chicago, 1918, p. 20.

is associated with acute pericarditis may be controlled, in large measure, by the application of an icebag to the precordia, care being taken that the bag is not too heavy. The treatment of the pain of angina pectoris has been described under that affection.

In milder cases, and in ambulatory patients, the above symptoms may often be ignored. The moderate use of bromides, or a few doses of veronal, is permissible. It is unwise to resort to the use of narcotics except in the more serious cases, and these latter require restriction to bed.

**DYSPNŒA.**—The use of extra pillows, or of a bed-rest, may serve to relieve much of the difficulty. Severe respiratory disturbance, due to effusion into the pericardium or pleural cavities, may require paracentesis, which has been described elsewhere.

When the dyspnœa is severe, and is associated with cyanosis, distention of the cervical veins, and well-marked increase in the cardiac dulness to the right, *i.e.*, when there is evidence of increased pressure in the large veins and enlargement of the right auricle, and especially when, in addition, the radial pulse is small and weak, venesection may be helpful. A large vein in the bend of the elbow is usually selected, and blood withdrawn until there is a lessening in the turgescence of the veins of the neck, and a decrease in the evidence of marked enlargement of the right chambers of the heart. The amount of blood may be 8 to 10 ounces, or two to three times that amount. In these cases it may be conceded that the dilatation of the heart is in excess of the physiologic limits, and the venesection acts by removing the factor of increased venous pressure. The resulting hydremia, or thinning, of the blood makes

easier the propulsion of the latter through the smaller vessels in the periphery. The blood volume is promptly restored, but the hydremia persists for days to weeks. This factor of hydremia cannot be produced by "bloodless venesection," *i.e.*, free purgation.

**INSOMNIA.**—This should be treated by the measures that are appropriate when it occurs in non-cardiac cases. It is highly desirable that patients suffering from marked heart failure obtain an adequate amount of sleep.

**GASTRIC DISTURBANCE.**—With relief of the heart failure, there may be a lessening in gastric symptoms. If gas is a prominent symptom, dietetic measures, such as restriction of carbohydrates, may be helpful. The remedies suggested under angina pectoris (*i.e.*, Hoffman's anodyne, etc.), may be tried. In addition, the methods used in the treatment of gastric disturbance presenting in patients not affected by heart disease are available. The relation of gastric symptoms to the action of digitalis will be described under that drug.

**EDEMA**, when present in cardiac disease, is to be treated by the administration of digitalis and diuretic drugs, to be discussed later. Since the reason that edema is a feature of one cardiac case with failure, and is absent in another, is not quite clear, the treatment of edema must be somewhat empirical. Reduction in the fluid intake, and the exclusion of salt from the diet, are indicated, but neither method is suitable for long periods. In intractable edema of severe degree, as a last resort, drainage may be accomplished by small incisions in the skin, or by Southeys capillary tubes. These last two procedures have the drawback that the limb must be dressed aseptically, in order

to prevent infection. Large blebs should be evacuated under aseptic conditions. For further consideration of the problem of persistent edema, the reader is referred to text-books on internal medicine.

**CARDIAC STANDSTILL.**—When this has occurred suddenly, it is sometimes possible to induce the heart to resume beating. In some cases of Adams-Stokes' syndrome, the heart action has been restored, apparently, in response to a vigorous slap on the precordia. Massage of the heart is the procedure required in most cases.

On cessation of the heart beats, artificial respiration by the usual methods should first be employed. If, after two to four minutes, no heart sounds can be heard, the abdomen should be opened by a high incision, and cardiac massage instituted. The operator's hand is passed into the abdomen, and up under the left lobe of the diaphragm, through which the heart may be felt. The heart is then compressed against the chest wall. This is done intermittently, and if spontaneous beats of the heart are felt, the massage should be interrupted for several beats, to allow the contractions to establish themselves. Continuous massage may cause the spontaneous contractions to cease. The compression of the heart is then continued for a short time and again interrupted. After the heart has resumed beating, it is safer to use too little than too much massage. The hand should not be withdrawn from the abdomen until the heart is beating well. During all this time, artificial respiration must be continued. Partial inversion of the patient will keep the cerebral centres bathed in blood, and assist somewhat in the filling of the heart.

To be successful, cardiac massage should be begun within five minutes of the onset of the standstill. Serious

damage to the brain and clotting of the blood in the right ventricle follow if the heart beats are absent much over fifteen minutes.

## DRUG TREATMENT

Many drugs have been administered in the therapy of heart affections, and but few of them are of real value.

DIGITALIS, or foxglove, is far and away the most important cardiac drug, and deserves, therefore, a somewhat fuller discussion.

It has been clearly established that the chief action of digitalis, in therapeutic dosage, is the production of a lessening of the conduction in the bridge of tissue connecting the auricles and ventricles. This heart-block is brought about by stimulation of the vagus nerve and by a direct action of the drug on the fibres of the bundle of His. The extent to which each factor is concerned in a given case varies with the individual patient.<sup>2</sup> Digitalis causes a shortening of the ventricular fibres, but opinions differ as to whether the drug also increases the contractile power of the heart. Clinical observations would certainly give an affirmative answer, but when the matter is investigated, it soon becomes apparent that it is extraordinarily difficult to obtain proof of scientific value. The slowing of the ventricular rate, due to the digitalis-produced heart-block, may account for much of the improvement in ventricular output.

The weight of recent evidence would appear to be against a constriction of the peripheral vessels due to digitalis. This lessens, if it does not remove, the contraindi-

<sup>2</sup> CUSHNEY, A. R., MARRIS, H. F., and SILBERBERG, M. D.: *Heart*, iv: 39, 1912-13.

cation to digitalis in conditions of hypertension. If edema is associated with cardiac failure, the use of digitalis will often be followed by diuresis.

The first and most constant sign of the action of digitalis is a depression of the T wave in the electrocardiogram. This appears at the same time as the beginning of a therapeutic effect, and has been demonstrated on animals to occur when 25 per cent. of the minimal lethal dose has been administered.

Digitalis also produces a lessening of conduction in the auricular tissues. When pushed, the drug may cause extrasystoles and finally ventricular fibrillation, with death. The occurrence of the extrasystoles is the explanation of the bigeminy or digitalis coupling; its presence is an indication for immediate discontinuance of the drug, as it has been shown<sup>3</sup> experimentally in cats, that when coupling (also extrasystoles and the phenomenon of ventricular escape) appears, 70 per cent. or more of the minimal lethal dose has been given.

Among the earliest of the symptoms of the toxic action of digitalis, is loss of appetite, then nausea and vomiting. That this is not due to a local action on the gastric mucosa is evident, since it has been demonstrated that even though the entire gastro-intestinal tract be removed from a cat, and digitalis be injected intravenously, the cat will go through the act of vomiting. Diarrhoea is an exceptional result of digitalis.

**PREPARATION.**—It makes little difference in what form digitalis is administered. Provided the preparation is potent and a sufficient amount is given, a digitalis effect

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<sup>3</sup> ROBINSON, G. C., and WILSON, F. N.: *Jour. Pharm. and Exper. Therap.*, 10: 491, 1917-18.

may be confidently expected. A well-known author writes: ". . . in my usage of digitalis, preparation plays no part, for I have long since learned that the powdered leaf made freshly into pills is as satisfactory a form of digitalis as either tincture or infusion, or, if the leaf is good, just as effective as digipuratum and digifolin, and far less expensive."<sup>4</sup> It should be remembered that the tincture rapidly loses potency if diluted with water.

Many American-grown digitalis leaves are as potent as the best of those produced in Europe. There is, however, no known method of growing or selecting the leaves, and no pharmaceutical procedure, that will ensure a preparation of known and unvarying strength. The only and reliable method of determining the potency of a given lot of digitalis, is that of testing the toxicity of the latter on cats or frogs in the laboratory. Preparations so tested are said to have been "standardized," and if reasonably fresh, so that much deterioration has not occurred, are so uniform in their potency, that their therapeutic dosage may be calculated quite accurately before their administration. It is not possible for individual druggists to conduct the biological tests necessary for the standardization of the preparations of digitalis which they dispense. When using these latter unstandardized products, the physician may be guided by their observed effects on previous patients. Consequently, it is the part of wisdom to use few preparations and obtain them from the same source, in order to predict with some degree of accuracy the proper dosage.

It has been shown that when digitalis is given in sufficient amount, a therapeutic effect may be observed in five

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<sup>4</sup> CHRISTIAN, H. A.: *Amer. Jour. Med. Sci.*, 157: 592, May, 1919.

to six hours, and the maximal action in twelve to eighteen hours. Pardee<sup>5</sup> found that, in the average individual, the body disposes of about 22 minims of the tincture per day, and it is necessary to replace this if it is desired to maintain the digitalization. In this connection it is well to remember that minims and drops are not the same; with many tinctures it takes two to three drops to equal one minim. It may be helpful to direct the patient to measure his dose in a minim graduate in preference to the drop method. In case it is difficult for the physician to induce a patient to take the larger doses, which seem indicated by recent experience, or if he himself is held back due to past habits of dosage, it is suggested that a change to a different preparation, such as the leaf pill, may make it easier to administer an increased amount. The danger of the cumulative action of digitalis has certainly been overemphasized.

**ADMINISTRATION.**—The “Eggles顿 Method” of dosage and administration has become a standard, and the writer cannot do better than to quote freely from Eggles頓's<sup>6</sup> summary of his method.

It is designed for rapid digitalization by oral administration. The amount of the digitalis to be used is expressed in terms of the activity of the drug, and the patient's body weight in pounds. The activity of the drug is determined by the cat method of Hatcher,<sup>7</sup> the unit being the weight of the dry drug, in milligrams, which is required to kill 1 kg. of cat when a solution is injected intravenously. This amount is called a *cat unit*. High-grade specimens

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<sup>5</sup> PARDEE, H. E. B.: *N. Y. Med. Jour.*, 110, 26: 1064, Dec. 27, 1919.

<sup>6</sup> EGGLESTON, CARY: *Jour. Amer. Med. Assn.*, 74, 11: 733, March 13, 1920.

<sup>7</sup> HATCHER, R. A., and BRODY, J. G.: *Amer. Jour. Pharm.*, 82: 360, 1910.

of digitalis, when not assayed by the cat unit, may be regarded as having an average activity of 100 mg. to the cat unit. The average total amount of digitalis required for oral administration to man is 0.15 of one cat unit per pound of body weight.

The calculation of the average total amount required by any given patient is accomplished as follows: The patient's weight (W) is determined in pounds, the cat unit (C.U.) of the digitalis is determined, and one of the following formulas is applied:

$$(I) \frac{C.U. \times 0.15 \times W}{1000} = \text{grams of powdered leaf in total amount.}$$

$$(II) \frac{C.U. \times 0.15 \times W}{100} = \text{cubic centimetres of tincture in total amount.}$$

$$(III) \frac{C.U.}{100} \times W = \text{cubic centimetres of infusion in total amount.}$$

In a patient weighing 150 pounds, and when using digitalis having an activity of 100 mg. to the cat unit, the three formulas will work out to 2.25 grams (34 grains) of the powdered leaf, 22.5 c.c. (3 V ss) of the tincture, and 150 c.c. (5 oz.) of the infusion.

The remainder of Eggleston's directions and comments will be quoted in full.

#### "ADMINISTRATION OF AVERAGE CALCULATED TOTAL AMOUNT."

(1). When the patient has received no digitalis within the preceding ten days.

a. In urgent cases.—From one-third to one-half of the total calculated amount is administered at the first dose. After an interval of six hours, from one-fifth to one-fourth of the total is administered. After a second six hours, from

one-eighth to one-sixth of the total is administered. Thereafter, if more digitalis is needed, about one-tenth of the total may be repeated every six hours until maximal digitalization is secured. In the case of the example given above with the total amount being 22.5 c.c. of tincture, the first dose would be 7 to 11 c.c., the second from 4 to 5 c.c., the third from 2.5 to 3.5 c.c., and thereafter about 2 c.c. every six hours if required.

*b.* Rapid, for non-urgent cases.—About one-fourth of the calculated total is to be given at each of the first two doses, six hours apart. Thereafter about one-tenth to one-eighth of the total is given every six hours.

(2.) When the patient has been taking digitalis within the preceding ten days.

Before further digitalis is prescribed, the patient is to be subjected to the most careful examination, including the use of polygraphic or electrocardiographic records if available, to determine whether or not there are evidences of digitalis action.

*a.* When evidences of digitalis action are absent.—The procedure is the same as outlined above, except that the total amount of digitalis required is to be reduced to 75 per cent. of the total calculated. Thus, in the example used, the total would be reduced to 17 c.c. instead of the calculated 22.5 c.c., and the fractions prescribed at each dose would be based on the former figure (17 c.c.). The usual one-tenth of the total every six hours may then be prescribed if necessary.

*b.* When evidences of partial digitalization are present.—It is best not to attempt to administer more than one-half of the total calculated amount of digitalis, divided

equally between the first three doses. In urgent cases in this group, however, one may administer 75 per cent. of the calculated amount, preferably in three equal doses, and then, if digitalization is not quite complete, one-tenth of the total amount may be prescribed every six hours.

**SAFEGUARDS.**—The appearance of one or more of the following criteria of adequate digitalization, or of minor digitalis intoxication, indicates the cessation of further administration, either permanently or temporarily.

(1) Nausea or vomiting (except when due to splanchnic congestion and present before treatment is begun).

(2) Fall of heart rate (not pulse rate) to or below 60 a minute.

(3) Appearance of frequent premature contractions, of definite heart-block, of marked phasic arrhythmia, or of coupled rhythm.

The observance of a six-hour interval between doses allows time for complete absorption of the preceding dose and the development of its full action on the heart, so that if the patient is examined just before the administration of each dose, dangerous intoxication can be absolutely prevented. In practice it is perfectly safe to give the first three doses without personally examining the patient before the second and third doses, if the one nursing the patient is properly instructed to look for nausea, vomiting, or slowing of the pulse to 60 or less a minute, before giving the succeeding dose, and to stop administration if any of these phenomena appear.

When a leaf, tincture, or infusion, the cat unit of which is unknown, is employed, 100 mg. may be taken as the cat unit; but not more than 75 per cent. of the calculated total amount should be given in the first three doses.

When the patient cannot be weighed, or when marked edema or general anasarca is present, the body weight (exclusive of edema fluid) must be estimated as closely as possible and the total amount of digitalis calculated as usual. Not more than 75 per cent. of the calculated amount should then be given in the first three doses."

Many modifications of the Eggleston method have been made. An easy rule to follow is that of 0.1 gram (1.5 grains) of the leaf to every ten pounds body weight of the patient. For digitan (American-made digipuratum) the same dose holds, while it is 1 c.c. (15 mm.), just ten times as much, for the tincture. For a 150-pound patient the dose would be 1.5 grams (22.5 grains) of the powdered leaf, and 15 c.c. (just under 3 ss) for the tincture. This is somewhat less than the amount advised by Eggleston, but experience shows that it is usually efficient and less often produces severe vomiting. A convenient method is to prescribe 2 pills<sup>8</sup> containing 0.1 gm. (1.5 grains) of the powdered leaf, or 2 c.c. (30 mm.) of the tincture *t.i.d.* for two to three days, and then to maintain the effect by one, two, or three pills (0.1 to 0.3 gm., or 1.5 to 4.5 gr.) of the leaf, or 1 to 3 c.c. (15 to 45 mm.) of the tincture, per day. The larger dose of the initial days should be reduced promptly if gastric symptoms appear. In cases in which actual vomiting results, the drug should be suspended until at least twenty-four hours have elapsed from the cessation of toxic effects.

It is important that the saturation with digitalis be maintained by the small daily dose, which is to be diminished or increased according to the indications in the indi-

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<sup>8</sup> It should be three if the pills contain .06 gm. (1 gr.) of the leaf.

vidual patients. By so doing, many patients may be enabled to continue at their work, and avoid the loss of time, with the damage to the health, that results from repeated attacks of severe heart failure. From an economic standpoint alone, this is an advance over the method of treating solely during the attacks.

By use of the above methods of the rapid administration of digitalis by mouth, it is rare that there is a true need of the introduction of the drug by another route. When there is special urgency, ampoules of digipuratum (digitan), or even the tincture of digitalis may be injected intramuscularly, or strophanthin may be given intravenously. The injections of digitan or of the tincture of digitalis should be given deep into the gluteal or lumbar muscles and the site massaged vigorously for several minutes. If given subcutaneously, or if any of the drug remains unabsorbed, it causes much irritation to the tissues and an abscess may result. The special preparations of digitalis are less reliable in their potency and do not contain all the active principles of the drug.<sup>9</sup>

*Strophanthus*, *squills*, *apocynum*, *convallaria*, *hellebore*, and other less well-known drugs, are members of the digitalis group that have had their advocates. None of them have any advantage over digitalis, and the results of recent work, such as that of P. D. White and his co-workers, demonstrate that these preparations are quite unreliable as regards their potency. *Apocynum* in therapeutic dosage, was attended with undue vomiting. *Strophanthus* is perhaps the best of the group. They might all with advantage be placed in the discard.

<sup>9</sup> WEISS, S., and HATCHER, R. A.: *Jour. Amer. Med. Assn.*, 76, 8: 510, Feb. 19, 1921.

**STROPHANTHIN** (Ouabain is practically the same), the active principle of strophanthus, is attractive in that it is available for intravenous injection in emergencies. It has the objection that its potency varies, and there is, therefore, greater danger of a sudden excessive digitalis action. If used, the average dose should not exceed 0.5 mg. ( $\frac{1}{120}$  gr.) of amorphous strophanthin, and 0.25 mg. ( $\frac{1}{240}$  gr.) of the crystalline form, and should not be repeated under some hours unless the patient is carefully reexamined. As stated above, digitalis, if properly administered, is nearly always preferable.

**CAFFEIN.**—Occasionally this drug may slow the heart by action on the extracardiac nerves. In animals, heart-block and ventricular extrasystoles have been produced. It is probable that, in moderate doses, caffeine improves the efficiency of the heart. T. S. Hart,<sup>10</sup> as a result of the observation of cases showing numerous extrasystoles, and of one case of a paroxysm of tachycardia after theocin, advises caution in the use of caffeine and allied drugs in myocardial conditions showing a high degree of irritability.

**ADRENALIN** is a failure as a circulatory stimulant. Its chief action is that of constriction of the peripheral vessels, and this, of course, does not facilitate the ready circulation of the blood. It should be employed with caution in anaesthesia, especially in that of chloroform, due to the tendency to produce extrasystoles and ventricular fibrillation<sup>11</sup> with fatal results. The limitation of hypersensitivity to adrenalin in the diagnosis of hyperthyroidism has been mentioned under that affection.

<sup>10</sup> Abnormal Myocardial Function, The Rebman Co., New York, 1917, p. 263.

<sup>11</sup> LEVY, A. G.: *Heart*, v: 299, 1914.

PITUITARY EXTRACT acts on the peripheral vessels, but has not as yet achieved any particular place in the treatment of heart disease.

THE NITRITES.—The value of these, especially of nitro-glycerine and amyl nitrite, has been presented under angina pectoris. There is no evidence of direct action on the heart, but the rate may be accelerated through reflex action on the vagus centre in the medulla. They are in no sense true cardiac stimulants.

AMMONIA may slow the heart rate by reflex nervous influences. The effect is transient. Ammonia is used in conditions of collapse mainly for its stimulant action on the central nervous system.

ALCOHOL, in small doses, causes local irritation and also acts reflexly on the circulation. The heart is never directly stimulated, but depressed, by large doses.

CAMPHOR is supposed to stimulate, directly, the heart muscle, but there is an absence of scientific evidence of beneficial influence. Stimulation of the centres in the medulla is the chief action.

STRYCHNINE acts directly on the nervous system and not directly on the heart. *Spartein* is neither a heart stimulant, nor a diuretic. *Barium chloride*, in small doses, has had its advocates, but the ready appearance of extrasystoles and ventricular fibrillation, noted by Levy,<sup>11</sup> should serve to exclude this drug. *Cactin* has been much advertised as a cardiac remedy, but the condition, functional heart disease, for which it is recommended, does better without drugs (see "Effort Syndrome"). Evidence is lacking that cactin has any beneficial effect on the heart.

**DIURETICS.**—Digitalis itself is a diuretic in cases in which edema is associated with heart failure. But certain drugs of the caffeine, theobromine group, are even more efficient for this purpose. Since they are to a certain extent irritant to the kidney it is good policy to administer them on alternate days rather than for many days in succession. It cannot be told in advance just which preparation will cause the diuresis, and therefore if one preparation is not successful, it is necessary to change to another, and so on until the diuresis is produced. It is not often that all of these preparations fail, if the edema is of cardiac origin. The occurrence of extrasystoles has been mentioned above, under caffeine.

Some of the more successful of these diuretic drugs, and their ordinary dosage, are: caffeine sodiobenzoate 0.2 gm. (3 gr.) *b.i.d.*, theobromin sodiosalicylate (diuretin) 0.3 to 0.5 gm. (5 to 7.5 gr.) 4 *i. d.*, and theophyllin sodioacetate 0.3 to 0.5 gm. (5 to 7.5 gr.) 2 to 4 *i. d.* Still another useful preparation is *theocin*, a synthetic theophylline, the latter being obtained from tea. Theocin sodioacetate is used in the same dosage as the theophylline salt.

**QUINIDINE SULPHATE**, a preparation from cinchona bark, is as yet in the experimental stage, but its promise is so great as to warrant mention. Quinidine slows conduction in the auricular tissue and prolongs the period in which the latter is refractory after a contraction. This is taken advantage of in the treatment of auricular fibrillation and flutter, in which the action of the drug may abolish the circus movement (see mechanism of these two arrhythmias). The prolongation of the refractory phase may

enable the contraction wave in the circus to close up the gap, and run into the area of tissue still in a refractory state, following the contraction in response to the wave in its previous circuit. This terminates the circus movement, and the impulses from the sino-auricular node again control the heart.

There are further effects of quinidine on the heart. The drug shortens the conduction between the auricle and ventricle by a paralyzing action on the vagus nerve, very much like that of atropine, and also causes a lengthening in the conduction by direct action on the bundle. These are opposing actions, the extent of each varying in different individuals. The paralyzing effect on the vagus accounts for the increase in the ventricular rate that is noted when quinidine therapy is employed.

The administration of quinidine in auricular fibrillation is proving successful in about one half the cases. Some of these relapse, however. The chances of success appear to be less if the fibrillation has been present for a long time and if the amount of failure of the heart is considerable; the result appears to be uninfluenced by the sex, age, presence or absence of a valve lesion, or character of the latter, etiological type of heart disease, etc. On the return of normal rhythm many patients experience marked relief, but in some no conspicuous benefit is evident. This is probably explainable on the ground that though the normal sino-auricular rhythm has been restored, extensive impairment of the heart may yet be present; quinidine sulphate cannot, of course, remove the lesions associated with rheumatic, syphilitic, or arteriosclerotic heart disease, etc.

It is obvious that the two effects of the drug in circus movement are opposing; if the effect of delay in

conduction in the auricular tissue preponderates, it is conceivable that the crest of the advancing wave may not be able to overtake the wave of refractory tissue in front of it, and the gap remains open, thus permitting the circus movement to continue. Also it is common for the action on the A-V conduction to cause an increase in the ventricular rate to 120 or 130, even in successfully treated cases, and this may force a discontinuance of the drug, because of the danger that the ventricle may suddenly develop an attack of tachycardia. The latter has occurred when the ventricle responds to all of the auricular contractions, as these are successively slowed by the drug to the region of 200. In an already damaged heart, such a ventricular rate is highly undesirable, though, fortunately, the effect wears off with a few hours of the discontinuance of the quinidine.

The present method of quinidine therapy is somewhat as follows. The heart is brought under the control of digitalis to relieve most of the heart failure and to offset some of the quinidine effects on the auriculo-ventricular conduction. Digitalis is omitted for a few days (about 3 to 5), to permit any excess to be eliminated. In the experience of the author it is well to commence the exhibition of quinidine before the ventricular rate has risen over 90, to lessen the height, if possible, of the increase in rate that may likely follow treatment. First, 0.2 gm. (3 gr.) of quinidine sulphate is given in capsules and repeated in two hours. If there are no untoward symptoms, it is probable that the patient is not hypersensitive to the drug, and the next day the real treatment may be instituted. This consists of the administration of 0.4 gm. (6 gr.) of quinidine sulphate 3 to 5 times per day for 3 to 5 days.



Courtesy of Frank B. Mallory

PLATE 5. Thrombi in the auricle. Note the corrugated surface of the large thrombus.



The drug has been given for a longer period without harm, but success is far more likely to occur within the first three days.

It is customary to discontinue the drug when normal rhythm is restored, and safety demands its prompt suspension if untoward symptoms appear. The symptoms of the toxic action of quinidine are essentially those of the better-known quinine. In brief these are: ringing in the ears, headache, nausea, vomiting, palpitation (tachycardia), delirium, etc. Serious respiratory paralysis was observed by von Frey in the original reports<sup>12</sup> on quinidine therapy. The author has encountered a single instance of respiratory paralysis. It occurred at the end of the second day of treatment in a patient in whom signs of toxicity of the quinidine had not previously been detected. Although recovery ensued, the condition was alarming. The maximum effect of the drug is present about two hours after it has been taken, and it wears away in large part in twelve to eighteen hours. Patients receiving quinidine therapy should be in bed, and seen sufficiently often by the physician to avoid the continuance of the drug after the appearance of toxic symptoms.

Disasters have occurred as a result of the throwing off of an embolus from the heart. While it is known that auricular thrombi are more common when fibrillation is present, and it seems reasonable that the restoration of coördinate contraction of the auricle may increase the chance of the discharge of an embolus, it should be clearly remembered that the latter event occurs in cases never given quinidine. However, the presence of hemiplegia, or the

<sup>12</sup> VON FREY, W.: *Berlin klin. Wochenschr.*, 55: 417, May 6, 1918, and 55: 819, Sept. 9, 1918.

history of the latter, if of probable embolic origin, should not be disregarded in deciding upon the pros and cons of the use of quinidine in an individual case.

It is emphasized that the administration of quinidine in auricular fibrillation has not yet passed the experimental stage. A confident prediction is made, however, that the use of the drug marks a real advance in cardiac therapeutics. Whether quinidine should be continued in small doses in order to prevent the relapse of a successfully treated case, is yet to be established. The literature on the use of quinidine is growing rapidly. The reader is referred to two particularly informing papers.<sup>13</sup>

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<sup>13</sup> DRURY, A. N., and ILIESCU, C. C.: *Brit. Med. Jour.*, 3170: 511, Oct. 1, 1921.

LEWIS, T., DRURY, A. N., ILIESCU, C. C., and WEDD, A. M.: *Brit. Med. Jour.*, 3170: 514, Oct. 1, 1921.

## APPENDIX

### ILLUSTRATIVE CASE REPORTS

INDIVIDUAL case histories often are particularly effective in impressing certain points. The following case reports have been selected as illustrating many of the conditions described in the preceding chapters. It has been necessary to limit the number of the histories, and in most instances the notes will be considerably abridged. It is better, as Sir Walter Scott said, in the preface to one of his novels, that the reader should occasionally be asked to step across a ditch than to wade through a morass of details.

The patients to be described were seen in the Heart Clinic at the Boston Dispensary, in the Boston City Hospital, or in consultation with private physicians. In many instances the means of identifying the exact origin of the case will be purposely omitted so that it may be permissible to discuss the case the more freely. The object sought is not, of course, adverse criticism of the work of others, but a frank discussion of the case.

### RHEUMATIC HEART DISEASE

CASE 1.—Hilda C., Boston Dispensary, No. 101,639. Married woman, twenty-eight years of age, housewife.

*Family History.*—Three children living and well. Two miscarriages at six weeks in the past two years.

*Past History.*—At age of thirteen, was told she had a leaking valve of the heart and later was informed that complete recovery had ensued. Denies rheumatic fever, growing pains, tonsillitis and chorea. Scarlet fever one year

ago. Has moderate shortness of breath whenever "run down." Occasionally has excessive flowing with menses.

*Present Illness.*—No symptoms. Not short of breath on ordinary exertion.

*Physical Examination.*—Thin and poorly developed; general stature that of visceroptosis. Many carious teeth.

*Heart.*—Impulse in the fifth space just outside left midclavicular line. Border of deep cardiac dulness, second to fifth space, is convex to left. Systolic murmur at apex almost masking the first sound. This murmur is transmitted towards base and to left axilla. No diastolic murmur appreciated nor elicited by exercise. Regular rhythm, rate eighty-two.

Wassermann reaction twice negative.

Röntgen mensuration of heart gave figures "which are within normal limits except that the measurement of the left auricle is a little larger in proportion than the other measurements. The findings suggest mitral disease."

*Treatment.*—Referred to dentist. Proper corset prescribed.

*Subsequent History.*—No diastolic murmur detected. No evidence of cardiac insufficiency. Patient satisfied that the corset is of benefit; is leading an essentially normal life.

*Diagnosis.*—Rheumatic heart disease, mitral involvement.

*Discussion.*—The character of the murmur at the apex, plus the evidence of cardiac enlargement (the region of the left auricle) as noted by physical and Röntgen examination, make it probable that this is a case of rheumatic heart disease. After observation repeated at intervals, my original opinion remains unchanged in spite of the absence of a definite history of a disease of the rheumatic group.

The situation at the age of thirteen is obscure and does not help us much. There is no evidence that the heart was changed by the attack of scarlet fever one year ago. The stature of the patient may be a sufficient cause for the occasional shortness of breath.

If nothing more happens to the patient's heart it is perhaps of little practical importance whether the diagnosis remain as at present or be changed to "Normal heart. Visceroptosis."

**CASE 2.**—The patient was a negro boy, sixteen years of age. Seen with family physician.

**Chief Complaint.**—Shortness of breath for one week, two days pain over precordia and in shoulders.

**Family History.**—Parents, three brothers, and two sisters are living and well. One sister died of pleurisy and "water round the heart;" one brother has heart disease following rheumatic fever and chorea.

**Past History.**—No scarlet fever, chorea, measles, pneumonia or typhoid. Mumps some years ago.

Rheumatic fever six years ago. Was ill, off and on, for eight months, being in bed two to three days at a time. The rheumatism went from joint to joint, recalls that ankles were visibly swollen at times. Was told that the heart was damaged. Has occasional attacks of tonsillitis but is never confined to bed because of same. During the past year has been well save for feeling of malaise for day at a time and averaging four to five times per month. Attributes this to gas in atmosphere (lives near gas works).

**Present Illness.**—One week ago noted considerable shortness of breath and has been in bed past five days. Had been horseback riding and is thought to have over-

exerted. Two days ago, severe pain appeared over heart and the anterior aspect of both shoulders. Cannot lie down flat the last two days. No definite chills or sweating, fever not noted.

Before this attack, the climbing of one flight of stairs caused dyspnoea and moderate precordial pain for two minutes. Walking on street caused no symptoms.

*Physical Examination.*—Sitting propped up with pillows, moderately orthopnoëic. Fairly well developed but thin. Two carious teeth, tonsils prominent, no enlargement of glands in the neck. Cervical veins not engorged.

*Heart.*—Diffuse impulse over the precordia and extending to sixth interspace almost to anterior axillary line. Apex impulse is ill-defined; timing of impulse over right ventricle and in midepigastrium shows it to be diastolic. Left border cardiac dulness is convex to left. To-and-fro friction rub audible over sternum and along right sternal margin. This rub is increased by pressure of the stethoscope and when patient assumes leaning forward posture. Systolic murmur maximum at apex and transmitted to base but not to neck. At the apex the first sound is audible with the systolic murmur, also the second sound, and following the latter a low-pitched, diminuendo diastolic murmur. Pulmonic second sound accentuated and louder than aortic second. Heart rate eighty-five, regular rhythm. Visible pulsation in eleventh and twelfth space below left scapula.

Blood pressure 90/50. Hemoglobin 80 per cent.

*Diagnosis.*—Rheumatic heart disease, active, heart failure considerable, mitral stenosis and regurgitation, acute pericarditis.

*Discussion.*—Obviously a chronic case (history, and presence of mitral stenosis) with an acute exacerbation.

It is not a purely mechanical disturbance of the heart but an active infection, as clearly shown by the pericarditis. The pain was of such severity as to be hardly compatible with an attack of simple heart failure, but was readily explainable by the detection of the pericarditis. The latter, however, is not always painful. The formation of adhesions may very likely follow this acute inflammation of the pericardial sac.

The history of rheumatic heart disease in a brother should be noted. It brings to mind the interesting observation that it is not uncommon to find this disease in more than one child of the same family.

The history is incomplete as there is no note as to the presence or absence of the pulsus paradoxus or of pressure signs at the base of the left lung. The condition was clear enough, however, without these data.

The case was treated as an active rheumatic infection and improved rapidly following the free use of sodium salicylate. The boy returned to school about three weeks later. Apparently the advice as to the desirability of tonsillectomy in the future has been disregarded by his parents.

**CASE 3.—A Hebrew tailor, thirty-nine years of age.**

*Chief Complaint.*—“Heart trouble.” Was told a year ago at a hospital clinic that he should return to work, but every time he does so, finds he has to quit after a couple of days.

*Family History.*—Wife, two sisters, and three brothers are alive and well. Wife has never been pregnant.

*Past History.*—Recalls no children’s diseases save measles. No chest trouble. Has never been particularly ill. “Rheumatism,” off and on, during the past ten years;

has never been confined to bed with it; only symptom was an indefinite pain in the legs. Occasional tonsillitis of mild degree. Denies venereal disease.

*Present Illness.*—While at work is liable to suddenly experience a sense of pressure in the general region of the left nipple, then feels as though couldn't breathe and as if he would choke. Has recently been told by the physician of his lodge that he has serious disease of a heart valve and that he must be very careful about exertion, for example,—“he must not even lean over to pick up a match.” Has to walk very slowly.

No cough, swelling of feet, or shortness of breath, save when excited (doesn't exert enough to know its effect on breath, etc.).

*Physical Examination.*—Fairly well developed and nourished. Tonsils large, teeth in excellent condition.

*Heart.*—Impulse visible and palpable in fifth space at nipple line; percussion of apex corresponds. The first sound at the apex is sharp and modified by a systolic murmur, the second sound is faint and followed by a low-pitched diastolic murmur which is diminuendo in type. The systolic murmur is transmitted to the base of the heart and to the axilla; the diastolic is audible over only a small area near the maximum impulse. Action regular, rate eighty-two. Blood pressure 140/90. Exercise test well tolerated; rate returned to eighty-four two minutes later.

*Diagnosis.*—Rheumatic heart disease, chronic, mitral stenosis and regurgitation.

*Later History.*—On a subsequent visit patient admitted that for twenty years he had had undue shortness of breath on exertion.

He was markedly hypochondriacal and several times ceased work because of a sensation of pins and needles all over his body, and sometimes a trembling of the arms and legs. By dint of considerable time and effort spent in explaining the situation and in reassuring him, he was enabled to overcome his fears of impending death and to regain control of himself. For two years he worked steadily at his trade, that of a stitcher in a tailoring shop. He learned to drive an automobile.

Twice at intervals of one year there was hemoptysis. The amount was only a few teaspoonfuls and all traces of blood disappeared in four or five days. There was no fever, and no tubercle bacilli in the sputum or other evidence of pulmonary tuberculosis. An electrocardiogram showed auricular hypertrophy (*i.e.*, P wave notched, duration 0.12 second, and  $3.5^{10^{-4}}$  volt). Occasional premature beats noted. No preponderance. Otherwise normal.

About two and a half years after this patient was seen for the first time, he reported he had suddenly become quite short of breath during an acute chest cold, with which there was much cough. The sensation of being choked up was attributed to the cold, but it persisted steadily. Examination showed the presence of absolute arrhythmia, rate of apex being one hundred and four, that at wrist ninety-six, making a deficit of eight beats per minute. Digitonin pills were prescribed, with such benefit that the patient has remained at work, and has little interest in entering the hospital to try the effect of quinidine therapy.

*Discussion.*—The history was essentially negative for the occurrence of a disease of etiological significance, unless one is satisfied with the indefinite "rheumatism"

described by the patient. The presence of mitral stenosis, however, justifies the diagnosis of the rheumatic type of heart disease.

Attention is drawn to the two attacks of hemoptysis. This is not uncommon in cases affected by mitral stenosis. It does not appear to have any evil prognostic significance.

The onset of auricular fibrillation at the time of some illness, as a chest cold, is not unusual. The appearance of occasional premature beats was the only suggestion that auricular fibrillation was more likely to ensue.

Undoubtedly the most important feature during the first part of the observation of this patient was the undue amount of apprehension. It cost me hours of psychotherapeutic conversation to undo what the previous physician had aided in causing by his pessimistic remarks. It was impressive also to note the amount of improvement in this patient after he had gotten to believe that his heart was still capable, with certain limitations, of doing its duty.

**CASE 4.**—Thomas M., Boston dispensary, No. 24,188. Twenty-three years old, electrician.

*Family History.*—No heart disease.

*Past History.*—Measles, whooping cough. At age of ten years, had chorea followed by heart trouble. Tonsillitis occasional. Tonsillectomy ten years ago. Denies venereal disease.

*Present Illness.*—Has been in Nose and Throat Clinic for treatment of a peritonsillar abscess. Referred for examination.

Is short of breath for about two minutes on ascending one flight of stairs. Can walk on street and do daily work without symptoms.

*Physical Examination.*—Remnants of tonsils visible.

Visible throbbing carotids, brachials, and other of the larger peripheral arteries.

**Heart.**—Cardiac impulse visible over a wide area in fifth and sixth spaces from inside nipple line nearly to midaxilla. Timing of cardiac impulse shows that it is mostly a systolic retraction. Slight systolic retraction below left scapula.

At the apex there is a systolic murmur transmitted to axilla and base. At aortic area there is another systolic murmur transmitted to carotid. Second aortic sound replaced by a loud, blowing diastolic murmur of greatest intensity along left sternal margin. At the apex the second heart sound is just audible, and following it is a low-pitched diastolic murmur. Regular rhythm, rate ninety.

Slight edema over tibiae. Blood pressure 145/40 (fourth phase). Traube's double sound heard over femoral; well-marked pistol-shot and Duroziez's sign over femoral and brachial arteries. Capillary pulsation present.

Two weeks later. Traube's sign absent.

Wassermann reaction negative.

**Diagnosis.**—Rheumatic heart disease. Aortic regurgitation and mitral stenosis.

**Discussion.**—Since there is evidence that the heart disease started at the age of ten years and followed chorea, it is practically certain that we are dealing with a case of rheumatic heart disease. Therefore, by all odds, the most likely interpretation of the apical findings is organic mitral stenosis and not a functional condition, *i.e.*, the condition described by Austin Flint. If there had been no previous intimation of heart disease, great care should be used before cardiovascular syphilis is ruled out.

The systolic retraction of the cardiac impulse is probable evidence of hypertrophy of the right ventricle; it, too, favors the diagnosis of organic change in the mitral valve. Regurgitation through the mitral valve is also present. The systolic retraction under the left scapula is doubtless the Broadbent's sign, and indicative of pericardial adhesions. The latter are common with the rheumatic type of heart disease and probably absent with the syphilitic.

I should not care to diagnose aortic stenosis in this case. Its presence would not essentially alter the prognosis or treatment. In fact, though this is a case of combined valvular lesions, it need not necessarily be of more unfavorable prognosis than one in which the mitral valve alone is affected.

The Traube sign in the femoral is a rare finding.

Tonsillectomy was advised. The recent occurrence of a peritonsillar abscess was noted in making this decision.

### SEPTIC HEART DISEASE

**CASE 5.**—Married woman, thirty-six years of age, born in Russia, a dressmaker.

*Chief Complaint.*—Pain in the left side for five days.

*Family History.*—Father died of asthma at age of eighty-four. Mother, four brothers, and three sisters are all alive and well; none dead. Husband died at age of thirty of heart disease.

Married at sixteen. Two miscarriages with a normal birth between. This child dead as the result of an accident.

*Past History.*—Mumps in childhood. No history of rheumatic fever, tonsillitis, chorea, scarlet fever, measles, whooping cough, pneumonia, or influenza.

Patient has known during the past fourteen years that

she had heart trouble. No dyspnoea, but following exertion, there is a sense of weakness so marked that she can hardly walk. There are frequent attacks of palpitation. One year ago swelling of the feet and legs was present for several weeks. Has been a bed patient in different Boston hospitals several times in the past twelve months. There has been no cough, sputum, or hemoptysis. No other symptoms. Has been a patient at an out-patient heart clinic for several months and is using pills of digitalis leaf daily.

*Present Illness.*—Yesterday had a definite chill, lasting twenty minutes. Attending physician is said to have stated that there was some fever. During the last four days there has been a more or less persistent pain in the lower left axilla.

*Physical Examination.*—Flush on cheeks, moderate cyanosis of lips. Apex impulse in fifth space at the left midclavicular line; it is accompanied by a slight thrill, systolic in time. At the apex there is a crescendo murmur ending in a sharp first heart sound; this is followed by the second sound, and after a just perceptible pause, by a diastolic roll. The latter is diminuendo in type. Pulmonic second sound accentuated and louder than the second sound at the aortic area. The rhythm is absolutely irregular; heart rate is seventy-five to eighty-five.

The record reads, "Presystolic murmur at apex ending in a sharp first sound." Later entries contain repeated notes such as, "Heart markedly irregular. Presystolic murmur at apex." The electrocardiogram showed auricular fibrillation. (It is reproduced in Fig. 22, the lower tracing.)

*Subsequent History.*—A few days after entrance quinidine therapy was instituted, but it had to be discontinued because of headache, increasing heart rate, and orthopnoea which appeared following the taking of but 0.4 gm. (6 gr.).

This patient remained in the hospital two and a half months. At times there were short attacks of vomiting. The most marked features were progressive weakness and emaciation associated with an irregular temperature, ranging from normal to a little over 102°. The remainder of the physical examination remained essentially unchanged.

Death ensued two and a half months after admission to the hospital.

*Diagnosis.*—Septic heart disease complicating chronic rheumatic heart disease, mitral stenosis.

*Discussion.*—The diagnosis of rheumatic heart disease and mitral stenosis seems evident from the character of the diastolic murmur at the apex. An apical murmur or roll of this description is pathognomonic of stenosis of the mitral valve, except when associated with evidence of aortic regurgitation.

For completeness the previous history should include the presence or absence of puerperal sepsis, and there might be some development of the statement, "Patient has known during the past fourteen years that she had heart trouble."

The diagnosis of septic heart disease seems most probable; no other cause was found to account for the chill at the onset of the present illness nor for the long-continued fever of irregular type. It is known that a septic infection of the heart is often engrafted on the lesions from a previous inflammation, especially that of rheumatic heart disease.

Since no blood cultures were attempted, the nature of the infecting organism remains in doubt. The record also lacks evidence of the discharge of emboli, unless the pain in the left side were due to a splenic infarct, but experience shows that this feature of septic heart disease may at times pass unnoticed.

Attention is drawn to the choice of the word "presystolic" for the crescendo murmur ending in the sharp first sound. At no time was there doubt as to the persistence of auricular fibrillation, but numerous observers described one of the murmurs at the apex as "presystolic." Since, when fibrillating, the auricles no longer exhibit a coördinate contraction, the latter could not, as is usually said, produce the murmur termed "presystolic" in this heart. The murmur is pre-sound but not pre-systolic, nor is it auriculo-systolic.

CASE 6.—A hotel steward, fifty-one years of age.

*Present Illness.*—Sudden onset of the sensation of weakness twenty-one days ago. Did not have a chill. The next day a cough with rusty, tenacious sputum set in. His physician reports that the patient has had a typical attack of lobar pneumonia of the right upper lobe. Said to have been desperately ill.

One week ago the temperature fell to normal by crisis and the patient was fever-free for the succeeding three days. Each afternoon of the past three days there has been a chill with severe shaking. The chill persists about one half an hour and is associated with a rise of temperature.

*Family History.*—Wife died of apoplexy; two children are living and well. Otherwise unimportant.

*Past History.*—General health good. No history of

disease of etiological significance as regards the heart. No precordial pain, dyspnoea, palpitation, or edema.

*Physical Examination.*—Looks slightly ill. Lungs. Slight dulness with broncho-vesicular breathing throughout the right upper lobe; over this area there are a few fine râles. There is slightly diminished breathing at both bases with some dulness.

*Heart.*—Apex impulse not seen or felt. Sounds distant, regular, and not rapid. The aortic second sound is greater than the pulmonic second; neither is accentuated. No murmurs are heard.

White blood count is 14,900.

*Subsequent History.*—The temperature was normal for two days after the patient's admission to the hospital and, the opinion was that the condition was that of a slowly resolving pneumonia; probably not empyema.

Five days later. Fever of an irregular type has been present during the past five days. The initial rise was accompanied by a chill and some pain in the right side, but, following the voiding of a large amount of urine, the temperature fell to normal that night. A few white cells and a few red cells were found in the urine. The question of a pyelo-nephritis is raised.

Ten days after admission. The white count is 15,000. There is nothing definite on physical examination or on X-ray of the lungs to bear out the suspicion of pus in the chest. Nothing abnormal found in the heart.

Fifteen days after entrance. The signs in the lungs remain unchanged save that there are no râles. Possibly this is a beginning tuberculosis, but the height of the white

count is against that diagnosis. Several 24-hour specimens of the urine have shown no pus and the kidney appears to have been eliminated as a possible source of trouble.

Twentieth day. There has been a distinct chill on each of the past three days. Blood smears are negative for the plasmodia of malaria. Two successive blood cultures have shown Pneumococcus, Type I. This evidence can hardly be neglected and makes the outlook distinctly poor. There is nothing in the physical examination or in repeated X-rays to suggest the possibility of empyema. Under these circumstances the focus must be in the heart.

Twenty-third day. The irregular temperature continues. Yesterday the patient complained of a sudden pain in the left side with some spitting of blood. This morning there are a few râles present low in the axilla. The suggestion here is that of a possible small embolus to the lung. Four doses of antipneumococccic serum, type one, have been given without noticeable improvement.

There is a friction rub in the left axilla, with moderate dulness and râles. The picture is not that of further consolidation. The heart sounds have not changed, although they are still very indistinct. The patient is losing ground day by day.

Thirtieth day. Last night the patient suddenly complained of a blurring of the vision in the left eye, which to-day is totally blind. In addition some thickness of speech has appeared. The patient is becoming irrational and the respiratory rate is accelerating.

Thirty-second day. Progressive weakening and death.  
*Autopsy*.—There is an infarct in the lower lobe of the

left lung. The area comprises about one third of the lobe and is roughly triangular in shape.

The pericardial sac contains thirty c.c. of fluid, in which flakes of fibrin are evident.

The heart weighs 325 gms. The tricuspid orifice is practically occluded by a large yellowish-white, friable, nodular vegetation, which extends along the entire base of one cusp. There are several smaller B-B shot vegetations on a second cusp. The larger vegetative mass is composed of a group of smaller nodules, the whole measuring about  $3 \times 3 \times 1.7$  cm.; it approximates the opposite heart wall. The right ventricle is lined with a non-adherent red blood clot about 0.4 cm. in thickness. There is no evidence of ulceration about the bases of the vegetations. The other valves are normal.

*Diagnosis.*—Septic heart disease (Pneumococcus, Type I), involvement of the tricuspid valve.

*Discussion.*—This case was carefully studied and it is interesting to note the various conditions that were suspected as the cause of the fever, thus, pyelo-nephritis, empyema, tuberculosis, and malaria. As is not unusual, the heart showed no changes pointing to the correct diagnosis. The blood found in the urine is a frequent occurrence in septic heart disease. The infarcts to the lungs and apparently to the brain furnished a strong clue. The diagnosis of septic heart disease often must be made from the presence of an irregular fever of unexplained origin, the evidence of the casting off of emboli, and confirmed, if possible, by the finding of the causative agent in blood cultures. It may happen, however, that the blood culture results in no growth.

## CARDIOVASCULAR SYPHILIS

**CASE 7.**—Married man, fifty-three years, real estate broker, seen in consultation with family physician.

*Family History.*—Wife and two sons living and well. Paternal grandmother and some of his mother's relatives suffered from asthma.

*Past History.*—Asthma began at two years and has been present, off and on, until fifteen years ago, when an operation on the nose brought relief. Pneumonia at thirteen and again at seventeen years. At the age of twenty, had a chancre with skin eruption, for which internal medication was taken for three years. Gonorrhea at twenty-seven, followed shortly by an arthritis, which confined him to bed for two months. No definite recurrence of the arthritis. Four years ago an inflammation of the eye appeared; this was diagnosed as syphilitic by a well-known eye specialist.

*Present Illness.*—Gradual onset of dyspnœa on exertion one and a half years ago. This increased so that eight weeks back he consulted his family physician. Marked edema of the legs noted the past ten days, and for four days he has been forced to remain sitting in a chair because of dyspnœa.

*Physical Examination.*—Orthopnoic, visible pulsation of the carotids, face pale, with slight cyanosis of lips, ears and hands.

*Heart.*—Impulse in fifth and sixth spaces, extending nearly to anterior axillary line. No thrill. By percussion the right border is three centimetres from midsternal line, and the left four centimetres outside nipple. No abnormal dulness at base of heart. Loud, blowing, diastolic murmur

audible from second right costal cartilage to apex, maximum along left sternal margin. A systolic murmur at aortic area and transmitted to neck; at apex there is a soft systolic murmur transmitted toward axilla and base. Second sound heard only at base of heart. Action regular. There is a slight impairment of resonance at the base of both lungs; with some medium râles. Pulses equal, rate fifty-four to sixty-four, definitely Corrigan in type. A faint capillary pulsation is present. Considerable soft edema of lower legs and in posterior thighs.

A slight improvement followed the restriction of the fluid intake and the free use of digitalis and diuretics, but death occurred ten days later.

*Diagnosis.*—Cardiovascular syphilis, heart failure of the congestive type, aortic regurgitation.

*Discussion.*—This patient was suffering from an enlarged aortic regurgitant heart with cardiac failure, but the diagnosis should not have rested there. The condition was probably an advanced stage of cardiac syphilis, and if, at an earlier date (the patient consulted a physician at the time of the eye affection, four years before), vigorous antisyphilitic therapy had been instituted, it is possible the process might have been checked before the integrity of the heart and aorta was seriously impaired. It is known that true bronchial asthma does not of itself damage the heart. The arthritis was probably of gonorrhreal origin, and if the same infection had attacked the heart, it would have done so by producing a form of septic heart disease, which, as is well known, is usually fatal within a relatively short period. Thus, having established the presence of aortic regurgitation, mitral stenosis, or what not, it is an error to-day not

to go further and endeavor to determine its etiology, a recognition of which may be of far greater importance.

CASE 8.—Boston City Hospital, No. 425,866. Man, fifty-three years.

*Family History.*—Wife living and well, never pregnant.

*Past History.*—Chancre admitted thirty-five years ago; treatment for nine months. Has had arsphenamine and mercury during the past five weeks. Otherwise recalls no previous illness until five years ago. Has lost fifty pounds in last two years.

*Present Illness.*—Five years ago was told he had a "bad heart." Since then has had occasional spells of shortness of breath and has been unable to work.

*Physical Examination.*—Visible pulsation of carotids and other peripheral arteries.

*Heart.*—Impulse in sixth space, almost at midaxilla. Soft systolic murmur at apex with first sound. At aortic area a diastolic murmur replaces the second sound and is well transmitted to left sternal margin. Rhythm is absolutely irregular.

Blood pressure 118/0, and later 128/0. Wassermann reaction strongly positive. The X-ray shows an enlarged heart and aorta. The electrocardiogram shows auricular fibrillation and left ventricular preponderance.

The auricular fibrillation was brought under control by digitalis and subsequently removed by the use of quinidine sulphate. Normal rhythm has persisted to date (three months).

*Diagnosis.*—Cardiovascular syphilis, auricular fibrillation, aortic regurgitation.

*Discussion.*—The above is similar to case No. 7, but apparently of slower progression. Attention is called to the presence of auricular fibrillation with a syphilitic heart, a combination which has been doubted by some. The response to quinidine therapy has been most helpful.

CASE 9.—Boston City Hospital, No. 424,996. Man, aged fifty years.

*Family History.*—Wife living and well; six children living and well. There have been no miscarriages.

*Past History.*—Frequent sore throat of moderate severity; no history of other infections of rheumatic group. Gonorrhea at eighteen; syphilis denied.

*Present Illness.*—The first symptom was dysphagia eighteen months ago. Palpitation, dyspnœa, and cough past sixteen months, during which time he has been unable to work. For two months has had frequent attacks of "smothering," most marked at night. Swelling of legs past week.

Previous entries to Hospital: June, 1920, the Wassermann reaction was negative on both blood and spinal fluid. The X-ray showed an enlargement of the aortic shadow suggesting aneurism. Diagnosis: aneurism of the aortic arch and aortic insufficiency. July, 1920, Wassermann reaction strongly positive.

*Physical Examination* (early in October, 1921). Left pupil larger than right, both react to light and distance. Slight cyanosis of face. Visible and palpable pulsation in suprasternal notch. Marked throbbing of right carotid; left neither visible nor palpable.

*Heart.*—Impulse in sixth space well outside nipple line. Supracardiac dulness at first space measures 8.5

centimetres. There is a loud systolic murmur over the base of the heart and it is well transmitted to neck. Diastolic murmur almost replacing second sound at aortic area and loudest along left sternal margin. At apex there is a systolic murmur with the first sound. Rhythm very irregular, averaging twenty-five per minute. Pulse at right wrist is well-marked Corrigan in type; that on left is barely palpable. Liver, lower border palpable five centimetres below costal margin in right mammary line, upper border at sixth rib. Marked edema of legs and external genitalia.

Röntgen examination demonstrates a fusiform enlargement of the transverse part of the aortic arch.

The electrocardiogram showed heart-block not quite complete, for the ventricular rate varied from a two to one to a one to one rhythm with the auricle. At times the additional "a" waves could be distinctly seen in the venous pulse of the neck during cardiac diastole.

The severe heart failure continued, and death followed early in October, 1921. A limited autopsy was obtained and disclosed a large aneurism of the transverse aortic arch, and this almost obliterated the openings of the left carotid and subclavian arteries. The aortic wall showed advanced syphilitic changes which were continued down to the aortic cusps. The heart was considerably enlarged; except for the fibrous thickening of the aortic valve, the valves were normal. Microscopic examination was not made.

*Diagnosis.*—Cardiovascular syphilis, partial heart-block, heart failure, aortic regurgitation, and aneurism of the transverse part of the aortic arch.

*Discussion.*—The symptoms of this aneurism began

eighteen months before death. The negative Wassermann reactions on both blood and spinal fluid in June, 1920, should be noted. Too many diagnoses of cardiovascular syphilis are discarded because of a negative Wassermann test. It is obvious from the description that this was an advanced case, and relatively hopeless to treat.

It is noteworthy that this patient was the father of six apparently healthy children. Perhaps undue emphasis has been placed upon the association of miscarriages and syphilis in the parents.

The pronounced systolic murmur at the aortic area and transmitted to the neck was proven not to be due to actual stenosis of the aortic valve.

The diagnosis of heart-block was easily made at the bedside by the plainly visible waves in the jugular vein when the heart (ventricular) rate was slow. That the block was not complete could be inferred from the changing rate, the latter sometimes being sixty or more per minute.

CASE 10.—Boston City Hospital, No. 429,730. Single man, forty-six years, member of fire department.

*Chief Complaint.*—Daily attacks of pain, starting in right lower jaw and radiating to right neck, arms, and precordia.

*Family History.*—Unimportant.

*Past History.*—Never sick except for accidents. In 1911, fall on spine; 1916, fracture of left tibia; 1917, fracture of left olecranon process; 1920, fracture of left clavicle, right humerus, sternum, and several ribs. Denies venereal disease by name and by symptoms.

*Present Illness.*—Since March, 1921 (eleven months ago), has had frequent attacks of pain which apparently

starts in right lower molar tooth and radiates to neck, shoulder, and arm on the right side, and to the upper precordia. Occasionally the pain radiates to the left shoulder and arm. These attacks follow exertion or the attempt to walk against a cold wind. The pain is severe enough to force the patient to discontinue whatever he is doing and remain motionless. Believes it would be a great effort to speak during the attack. The latter lasts for only about two to five minutes. Is conscious of a distinct lessening of physical power since the attacks began eleven months ago.

*Physical Examination.*—Teeth appear normal; no tenderness to pressure on right lower molar.

*Heart.*—Increased supraventricular dulness especially to the right at first intercostal space. Soft systolic murmur at aortic area and transmitted to neck. Blood pressure 104/76.

X-ray of teeth shows no pathology. The chest plates show a prominence of the aortic shadow to the right. Width of great vessels six centimetres. The cardiac apex is lowered into the left curve of the diaphragm, and the outline of the lateral wall of the left ventricle is lengthened. Transverse diameter of heart sixteen centimetres.

The Wassermann reaction on two occasions was moderately positive.

The electrocardiogram was normal.

*Diagnosis.*—Probably cardiovascular syphilis, angina pectoris.

*Discussion.*—A case of angina pectoris, and at the patient's age, forty-six years, should first suggest syphilis as the cause. It is true that the point of origin of the pain

is atypical, but such occurs and has been described by Sir James Mackenzie in his book on heart disease. The association of the pain with exertion and the other data pertaining to the attacks are of considerable supporting value to the diagnosis. The history of the numerous injuries is somewhat confusing, but should not, it would seem, prevent the diagnosis of angina pectoris, probably of luetic origin. The X-ray and Wassermann lend some support to this diagnosis. It is extremely important that a therapeutic test of antisyphilitic treatment be instituted without delay. The patient was discharged to his private physician with this advice.

### ARTERIOSCLEROTIC HEART DISEASE

**CASE 11.**—Married woman, aged seventy years.

**Complaint.**—A few months ago was told by family physician that she had valvular heart disease and must be very careful about physical exertion.

**Past History.**—Measles at the age of twenty-two years. Is positive she has had no other diseases. Health has been excellent, save for some pain in the feet and lower back, for which she has been under the care of an orthopedist.

**Present Illness.**—A few months ago was told of the presence of a murmur indicating a leaky valve of the heart. Since then has noted that she must take her time on ascending stairs. Walks considerably but is a little short of breath on ascending hills. At times is conscious of a slight aching under the right breast; this is not a real pain and is never felt higher up over the precordia, and there is no radiation to the arms or shoulders. At times the heart pounds a

little (indicates by motion of hand a rate estimated at about one hundred per minute).

*Physical Examination.*—A fairly well-developed woman, somewhat thin; looks ten years less than her stated age. Arcus senilis present. Some of molar teeth absent, remainder in excellent condition.

*Heart.*—Impulse in fifth space at left midclavicular line. A slight systolic thrill is present. Percussion borders of heart normal, no increase in supracaardiac dulness. Regular rhythm, rate seventy-two. Loud, rough systolic murmur with first sound at apex, transmitted toward base. A faint systolic murmur at the second right costal cartilage and propagated slightly toward carotid. No diastolic murmur heard. Light exercise well tolerated, no essential change in heart findings. Blood pressure 160/95.

*Diagnosis.*—Arteriosclerotic heart disease.

*Discussion.*—The murmur at the apex was doubtless due to the regurgitation of blood through the mitral valve, and the latter may have undergone some of the changes found in arteriosclerosis, but it is doubtful if any good purpose would be served by adding mitral regurgitation to the diagnosis. It seems better to restrict this term to cases in which there is also evidence of cardiac enlargement and a history of infection of the rheumatic type. The picture associated with a "leaky valve" was not truly present.

Also heart failure can hardly be said to be present, as this lady could do fully as much if not more than the average for her age and type. The diagnosis of valvular heart disease was based apparently on the auscultatory findings; a systolic murmur without other evidence of abnormality

is insufficient grounds for the diagnosis of an organic change in the valve. The age of seventy years may be said to justify placing this patient in the arteriosclerotic group and by so doing the problems of prognosis and treatment are clarified.

CASE 12.—Retired business man, aged seventy-one.

*Chief Complaint.*—Severe attacks of substernal pain past six months.

*Family History.*—Unimportant.

*Past History.*—Good health until recent years. Appendectomy five years ago. Since then has had some lack of control of the bladder; told it is due to an enlarged prostate gland. Three years ago had pneumonia; was in bed two weeks. One year ago "rheumatism," evidenced by pain in left upper arm. This pain was not associated with exertion, and was of but a few minutes' duration at a time.

*Present Illness.*—Six months ago, while walking hurriedly, was seized by pain in throat (just below larynx). On stopping a few minutes the pain ceased. About three days later had a similar attack on ascending stairs. Now the attacks occur daily and sometimes several times per day.

The present attacks occur mostly in the evening and may follow even the exertion of undressing. The chief pain is under the midsternum, and may radiate to either upper arm, the left forearm, and occasionally to both middle fingers. The pain is severe enough to bring tears to the patient's eyes, and when it is present he must cease speaking and keep motionless. During end of attack the patient expels considerable gas from stomach. Has been given one and two drop doses of nitroglycerine but has not noted any effect.

Tobacco and alcohol none. No coffee for several months. Bowels always costive.

*Physical Examination.*—Thin, nervous man, looks his age. Pupils equal and react to light and distance; arcus senilis present. Three carious teeth.

*Heart.*—Supracardiac dulness four by three centimetres, palpable pulsation in suprasternal notch. Cardiac impulse faintly palpable in fifth space just inside left mid-clavicular line; left cardiac border inside nipple line. Heart sounds of fair quality, the normal physiological difference between the first and second sound at the apex is lessened. At the aortic area there is a faint systolic murmur transmitted slightly toward the right neck. Heart rate eighty-two, regular rhythm.

Knee jerks present and equal. No edema of legs.

Blood pressure 145/90.

Urine clear, slightly pale, specific gravity 1008, no albumen, no sugar.

Röntgen examination. Heart slightly small in size. Prominence of aortic knob and "tortuous" appearance to aorta.

*Diagnosis.*—Arteriosclerotic heart disease, angina pectoris.

This patient obtained "wonderful relief" from the use of nitroglycerine (tablet of  $\frac{1}{100}$  grain under the tongue) and a considerable lessening of the gastric flatus after taking Hoffman's anodyne (as described in Chapter XVI). However, he never regained much physical power. At times digitalis was prescribed but without marked benefit. The angina was promptly controlled and never returned with any severity. Death ensued some eighteen months

later, the last month being spent in bed and the picture being that of old age and moderate heart failure.

*Discussion.*—As is not uncommon, the relief of the symptoms of angina pectoris was followed by abundant evidence that the patient's heart was insufficient; in a word, he became a chronic invalid. Experience has shown that little else can be expected when angina pectoris appears in the elderly.

The success obtained by the administration of nitro-glycerine, in spite of its failure when previously prescribed, should be noted. In my opinion the method and preparation were less favorable. It is also suggested that the particular solution may have lost some of its potency, no effect being observed. The drug should be used in increasing dosage until relief or toxic symptoms result.

### HYPERTENSIVE HEART DISEASE

CASE 13.—Housewife, forty-two years of age, born in England. Boston Dispensary, No. 183,822.

*Family History.*—Father and mother died of old age. One sister and one brother are alive and well. Husband in good health. No children, no miscarriages.

*Past History.*—Measles and mumps in childhood. Influenza three years ago, erysipelas two years past.

*Present Illness.*—For two years has had much frontal headache. Urination frequent at night. Gradual lessening of strength. Occasional puffiness of ankles, face, and hands. Repeated urinary examinations have shown a specific gravity of 1008 to 1012, with albumen varying from a slight trace to a trace. The sediment has usually been free of pathological elements, but rarely has shown a hyaline granular cast.

The Wassermann test has been negative. The blood examination showed hemoglobin of sixty-five per cent., with three million red cells. The differential count of the white corpuscles, and the appearance of the red cells in the stained smear were essentially normal.

Has been treated by diet, etc., with indifferent success. Sent to Heart Clinic for consultation.

*Physical Examination.*—Considerable pallor; patient looks sick.

*Heart.*—Impulse in fifth space, almost at anterior axillary line. Percussion suggests enlarged heart. At the apex there is a loud systolic murmur with the first sound, and transmitted toward base and axilla. Regular rhythm, rate eighty-four. Blood pressure 175/126.

This patient was referred to the Massachusetts General Hospital, where she died eleven days later. The diagnosis was: chronic nephritis, uremia.

*Discussion.*—It was recognized that this patient was losing ground in spite of almost two years of treatment as an outpatient. The anaemia, and the comparatively fixed specific gravity of the urine, with the other data, pointed to damage to the kidneys. The belief that a serious condition was present suggested the desirability of recommending the patient for admission to a hospital in order that a more thorough investigation of the condition of the kidneys might be made.

Some would prefer to classify this case simply as chronic nephritis, but from the standpoint of cardiology it belongs in the group of hypertensive heart disease with chronic nephritis.

**CASE 14.**—Male printer, aged fifty-one years. Seen in consultation with family physician.

*Present Illness.*—Four days ago, when first seen by physician, appeared to be about "all in;" was dusky, dyspnoëic and staggering in gait. Sent to bed at once. Has had no fever nor clear-cut cough, but there have been pulmonary signs suggesting pneumonia, and some evidence of heart failure.

No pain near heart and none in arms or shoulders. Admits that there has been considerable shortness of breath for three months, following some heavy lifting. During the past two weeks has spent considerable time at night sitting in a chair, as breathing has been easier thus.

*Past History.*—Two years ago the blood pressure was about one hundred and eighty, and has been high since then. Has worked hard but has enjoyed fair health. Further history not obtained (in part because of patient's poor condition).

*Physical Examination.*—Markedly orthopnœic, slightly cyanotic. Chest deformed (pigeon breast, scoliosis, and dorsal kyphosis). Coarse moist râles in both axillæ. Impairment of resonance at right apex from second rib in front to spine of scapula behind. Over this area the breath sounds are weakened. High up in the right axilla there is a broncho-vesicular respiration and some râles of a crepitant type (not quite the fine crepitant râles of Laennec).

*Heart.*—Impulse in fifth space outside nipple line. No increased supraventricular dulness. Due to deformity of chest, it is hard to estimate the size of the heart by percussion, but by comparison of its contour with the stature of the patient, it would appear to be enlarged. Absolute arrhythmia present; rate at apex one hundred and twenty-six, at wrist one hundred and five, pulse deficit twenty-one.

Cervical veins engorged, no auricular wave visible on inspection. Second sounds present at base, systolic murmur at apex.

**Diagnosis.**—Auricular fibrillation in a heart that is probably hypertensive. Pulmonary condition uncertain; probably beginning consolidation of right upper lobe, but the findings may be due to the chest deformity plus the râles associated with severe heart failure.

**Subsequent History.**—The free use of digitalis was followed by a satisfactory control of the auricular fibrillation, the heart rate dropping to eighty to ninety with practically no deficit. The râles disappeared from the chest, but otherwise the physical signs in the right axilla remained unchanged. Fever remained absent and there was no further evidence of pneumonia.

About a month later, after the patient was up and about the house daily, his wife was awakened early one morning by noting that the patient was breathing with difficulty. She arose to get him a glass of water and when she returned he was dead.

**Discussion.**—In this case the presence of auricular fibrillation and the need for its control by digitalization were evident. There was much uncertainty of the pulmonary condition. Treatment was therefore undertaken for the relief of the heart failure.

The sudden death was quite unexpected. Although the attending physician signed "cerebral hemorrhage" on the death certificate, it is clear that at least two other possibilities must be considered. The discharge of an embolus from the heart to the lungs or brain may have taken place. A clotting of the blood, particularly in the auricular append-

ages, is not uncommon when the auricles are fibrillating. A rare possibility as a cause of this death is, perhaps, the onset of fibrillation in the ventricles. It has been noted experimentally, in animals, that sometimes when the auricles are fibrillating, the same mechanism may appear in the ventricles. Such an event probably occurs occasionally in human beings. In the absence of an autopsy the actual cause of death must remain unsettled.

The diagnosis of hypertensive heart disease seems most probable from the two-year history of high blood pressure, the apparent hypertrophy of the heart, and the absence of the history or physical findings pointing to some other etiological type of heart disease. Finley<sup>1</sup> has reported heart changes as a result of a deformity of the spine of a considerable degree. In this case the condition of hypertension seems a sufficient cause, and I doubt the influence of the spinal deformity as a factor in this case. It is probable, however, that the pulmonary findings are explainable in part, at least, by the deformity of the chest.

### THE HEART IN HYPERTHYROIDISM

CASE 15.—Housewife, aged forty-nine, born in Russia.

*Chief Complaint.*—Nervousness.

*Past History.*—Denies previous disease. Good health until four years ago, when home conditions became intolerable. Husband became very abusive. After a few months the patient became nervous, and would jump at the slightest noise. Was advised at Psychopathic Hospital to take baths. Has done this for three years with

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<sup>1</sup> FINLEY, F. G.: "Spinal Deformity as a Cause of Hypertrophy and Dilatation," *Canad. Med. Jour.*, xi, 10:79, Oct., 1921.

apparent benefit, but always notes the return of the nervous feeling soon after reentering her home.

Was a patient in the hospital two months ago. Complaint then was that the heart had been "jumping" for three months and there was dyspnoea and palpitation on exertion. She was very nervous and tired. The catamenia had ceased about two months.

The physical examination during this stay in the hospital was essentially normal save for a thrusting apex beat accompanied by a systolic thrill. The blood pressure was 190/89, and the pulse rate eighty to one hundred. The electrocardiogram showed sinus arrhythmia and a rate of one hundred and fifteen to one hundred and twenty-five. On discharge the diagnosis was: menopause, hypertension.

*Present Illness.*—Since leaving the hospital five weeks ago the nervousness has increased. Also the dyspnoea and palpitation on moderate exertion, such as walking up one flight of stairs, has increased. One month ago a dry cough and swelling of the ankles appeared.

Patient noted a swelling in the neck (region of the thyroid) about six months ago. Perspiration is very free. There has been a loss of twenty-five pounds' weight in the past year. The monthly periods are still absent, now four months.

*Physical Examination.*—Thin, very restless woman, mentally clear. The eyes show no signs of Graves's disease. The thyroid gland is readily visible and palpable, and over it there may be heard a bruit. There is a visible pulsation of the carotid arteries. Well-marked fine tremor of hands. Skin warm and moist.

*Heart.*—Heaving impulse in fifth space one centimetre

outside left midclavicular line. By percussion left border is about 0.5 centimetre further to left; right border measures 3.5 centimetres from the midsternum. No thrill. Action rapid and irregular. There is a systolic murmur loudest at the apex and transmitted over the precordia.

The lungs are clear. There is slight soft edema of the ankles. Blood pressure measures 145/90.

The electrocardiogram shows auricular fibrillation with a ventricular rate of one hundred and sixty-five to one hundred and eighty-five.

After a few days' administration of digitalis, a second electrocardiogram showed the same mechanism at a rate of one hundred and twenty-five to one hundred and thirty-five and a flattening of the T wave.

The basal metabolism was 61.7 per cent., and after a few days a second test showed a rate of 61.2 per cent.

*Diagnosis.*—Hyperthyroidism, auricular fibrillation.

*Discussion.*—During the first stay in the hospital it was noted that at times the heart action was rapid and irregular and then a more normal rhythm returned. The first electrocardiogram was not taken during the rapid rhythm. It is possible that what the interne described were transient attacks of auricular fibrillation. The latter has apparently become fixed.

In view of the later findings and the height of the basal metabolism on two occasions, the diagnosis of hyperthyroidism would seem clear. Otherwise the case would be one of auricular fibrillation of unexplained origin. The lowering of the blood pressure after the establishment of the fibrillation is to be expected; also the record is less accurate than when normal rhythm obtains.

## THE HEART IN DIPHTHERIA

CASE 16.—Boy, eleven years of age. Admitted to the South Department of the Boston City Hospital on October 9, 1921. Diagnosis,—faucial diphtheria. October 10, profuse watery muco-gelatinous discharge from right nostril, membrane lining right nasal cavity; left side clear, no discharge. Throat entirely filled with edema and membrane. Tonsils completely covered with a grayish-white raised membrane, which extends  $\frac{1}{2}$  centimetre on to anterior pillars and on to soft palate on left for distance of about two centimetres. The posterior pharyngeal wall is completely obscured. Breathing through the nose is entirely prevented. There is a marked edema of neck, more marked on left side; patient very toxic.

The heart sounds are of fairly good quality.

October 11.—Slight improvement in membrane, edema a little less. Heart sounds and pulse are somewhat weakened. Patient less toxic. Epistaxis for five minutes with a spontaneous cessation.

October 12.—Membrane rolling up. Patient less toxic. Suggestion of presystolic roughening; regular rhythm.

October 13.—Process is subsiding very definitely. Splitting of first heart sound but no suggestion of a presystolic roll.

October 16.—Normal temperature. Membrane nearly all gone. Edema of neck now much less. Heart sounds good.

October 20.—Throat no longer sore, edema of neck gone. Heart sounds of good quality, no evidence of cardiac enlargement.

Patient vomited this A.M.

October 21.—Patient was very uncomfortable last night and had a weak, irregular pulse. This morning the heart sounds are regular and of good quality but vary frequently.

General condition good. No evidence of palatal paralysis; knee jerks present.

October 23.—Blood pressure better. No evidence of paralysis. The heart sounds are weaker with an occasional extrasystole.

October 25.—Heart regular, no splitting of first sound or extrasystoles.

October 31.—Heart 3.5 by 8.5 centimetres. Action is very irregular, at times without any regular sequence of rhythm, occasionally a few extrasystoles, and again a nearly normal rhythm. Four-sound gallop made out several times.

November 1.—Heart slightly enlarged, absolute irregularity of the beats. At times there is a splitting of the first sound and a typical gallop rhythm, and at other times four beats.

November 3.—Heart is absolutely irregular, rate 140 to 160. Diffuse apex impulse in fourth and fifth spaces just outside nipple line. Tenderness over the liver and in the epigastrium. Lips are slightly cyanotic; the extremities moist.

Examination shows a fairly strong but varying impulse in fourth and fifth spaces just inside the nipple line. Area of dulness not made out as enlarged.

Great irregularity in strength and rhythm of heart sounds, absolute irregularity but with periods, estimated as part of a second, during which there is a series of rapid regular beats, and at times a semblance of gallop rhythm.

Patient's color is very pale, the lips are ashy and at times slightly cyanotic. Skin cool. Seven-thirty P.M.—Vomited again. At times the pulse pressure is only eight millimetres of mercury, but now is twenty-six millimetres.

November 5.—Cheyne-Stokes respiration present last night and this morning. The gallop rhythm is still present but less marked and the heart sounds are stronger. Vomiting is occurring three to five times per day.

November 7.—Condition about same, heart more regular, now has a loud systolic murmur over precordia. There are some moist râles at base of the lung. Patient is not vomiting but has received nothing but small amounts of water.

November 9.—Better. Doesn't complain of precordial or epigastric distress.

The heart sounds are somewhat irregular but of good quality. Fewer râles at the pulmonary bases; respiration normal. Pulses of very low tension.

November 13.—Continues to improve. The heart action has gradually become regular and the sounds are of good quality. No further vomiting. Sleeps fairly well without morphia.

November 14.—Regular rhythm with typical four sound gallop. No evidence of cardiac enlargement. Liver edge just below costal margin.

November 18.—No murmurs over heart. Occasional extrasystoles. Knee jerks absent. There is considerable emaciation of the tissues.

November 22.—Heart sounds normal. Pulse of fair tension.

December 16.—Up in chair for one hour.

December 19.—Abscess on thigh drained. This is apparently the site of former hypodermic injection. The culture showed no growth.

December 26.—The knee jerks have returned slightly.

January 13.—Up and about. Heart appears normal. Patient is still thin and somewhat weak. The heart rate accelerates with exercise but promptly returns to normal. Discharged from hospital.

May 2.—Patient returned for examination.

*Heart.*—Apex in fifth space just inside nipple line. Sounds clear, no murmurs, action regular. Rate—at rest, eighty; after hopping fifteen feet, ninety-six; one minute afterward, seventy-two; after hopping thirty feet, eighty; one minute afterward, sixty-eight. Tires easily.

The electrocardiogram, on January 26, was normal in all respects.

*Diagnosis.*—Faucial and nasal diphtheria, severe toxic disturbance of the heart, recovery.

*Discussion.*—I am indebted to Dr. E. H. Place for the above case history, which I have abstracted somewhat fully, as it offers a striking picture of the functional disturbances which may be met in severe diphtheria. Electrocardiographic records were not procurable until the patient was fully convalescent, but the bedside notes are quite satisfactory.

This patient was very toxic at the time of admission, and in view of the extensive amount of diphtheritic membrane that was present, it could confidently be expected that the heart would later show serious disturbance. During the first three weeks the cardiac findings were those of a minor type, as changes in the first heart sound, variations

in rate, extrasystoles, etc. By October 31, an inconstant gallop rhythm was noted. On the third of November the description of the rhythm is that of auricular fibrillation. The low pulse pressure, at one time measuring but eight millimetres of mercury, is noteworthy. In early November the gallop rhythm was present much of the time for several days. This rhythm, as occurring in diphtheria, is nearly always due to heart-block.

The result of the exercise test performed in May, nearly three months after the patient's discharge from the hospital, is interesting; as is also the normal electrocardiogram taken on January 26. These points conform to the rule that if the patient survives he ultimately makes a complete clinical recovery.<sup>2</sup>

## CONGENITAL HEART DISEASE

**CASE 17.**—A baby dying on the ninth day.

**History.**—Weighed seven pounds and ten ounces at birth. Delivered by the operation of intermediate forceps; pituitrin was not used. Was not a blue baby. Nursed irregularly, well at some feedings and poorly at others; inclined to be drowsy and cried little. Definite cyanosis was never present, but there was a slight tendency to cyanosis when the baby cried. On the fifth day there was quite marked jaundice, which had cleared in part at the time of death. The heart appeared normal on examination.

At the 6 P.M. nursing period on the eighth day, the baby took the breast poorly. At 12 P.M., respiratory difficulty and cyanosis were noted. Death occurred at 5 A.M. of the ninth day.

**Family History.**—Mother, aged thirty-five years, in

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<sup>2</sup>Exception to this has been mentioned in chapter xiii.

good health, married seven years. No other children. There was one miscarriage, cause unknown, at two months, about five years ago. At the sixth week of this pregnancy, miscarriage was threatened by slight flowing for ten days. There was considerable morning sickness; the pregnancy then proceeded normally. Father, aged thirty-five, in good health.

*Autopsy.* *Heart.*—The right side is large and full of blood, while the left cavities are almost empty. The right ventricular wall is distinctly thicker than the left. The upper third of the interventricular septum is absent, and the opening thus formed is continuous with that of the foramen ovale between the auricles. The annulus from the posterior wall of the left auricle is present but is not adherent to the margin of the foramen ovale. The mitral and tricuspid valve flaps are almost in contact as they descend into the ventricles. The ductus arteriosus is freely patent. There is a constriction, amounting almost to obliteration, in the aorta at a point one half inch before it is joined by the ductus arteriosus.

The liver shows beginning central necrosis.

There is a blind pouch in the small intestine leading to the umbilicus. This is probably a Meckel's diverticulum.

*Diagnosis.*—Congenital heart disease, patent interventricular septum, patent ductus arteriosus, coarctation of the aorta, beginning central necrosis of the liver.

*Discussion.*—The unexpected death of this infant was not explained until the autopsy had been performed. Otherwise it might have been thought to have been a delayed result from the operative delivery. Emphasis is laid upon the extensive abnormality in the heart and

adjacent vessels without the presence of evidence sufficient to make possible the diagnosis before death.

The greater thickness of the right ventricular wall is the rule at birth. Also, since the ductus arteriosus was still freely patent, it was not surprising that there was no evidence of the adhesion of the annulus to the margin of the foramen ovale. The constriction of the aorta was probably unimportant, as the blood would doubtless have been conveyed by the development of collateral vessels. The change in the liver cells is most likely due to the imperfect arterial blood which must have resulted from the abnormalities in the heart and vessels.

Whether the threatened miscarriage at the sixth week had any influence in the failure of development of the interventricular septum is, of course, an open question. It is noteworthy that the aortic part of the septum should have been forming at about this time and the septum should have been complete before the end of the third month.

**CASE 18.**—Twenty-year-old girl, single.

**Family History.**—Father and mother dead, cause unknown. One sister died at the age of twenty-four of pulmonary tuberculosis. Another sister is living and well.

**Past History.**—Scarlet fever when child; otherwise recalls no illness. Could run and play with perfect freedom.

**Present Illness.**—When undergoing a physical examination preliminary to the acceptance of employment with the United Fruit Company, an abnormality in the position of the heart was discovered. There are no symptoms of disease or departure from normal health.

**Physical Examination.**—Well developed and nourished,

*Heart.*—Impulse in fifth space just inside right mid-clavicular line. By percussion heart is located to right rather than left of sternum; right border of heart is 7.5 centimetres to right of midsternum and left is 3.5 to left. Liver dulness apparently present on left side from sixth rib to costal margin, where its lower edge is just palpable. Examination otherwise normal.

Röntgen examination shows transposition of thoracic viscera.

The electrocardiogram is normal save for an inversion of all waves in lead I.

*Diagnosis.*—Congenital heart disease, dextrocardia.

*Discussion.*—The abnormal position of the heart is but part of a general transposition of the viscera. The inversion of all the waves in lead I of the electrocardiogram is diagnostic of congenital dextrocardia and is a differential point from dextrocardia acquired after birth. The condition of congenital dextrocardia is compatible with health.

## EFFORT SYNDROME: IRRITABLE HEART

CASE 19.—A returned soldier, aged thirty years.

*Past History.*—Excellent health. Recalls no illness until time of army service. Was gassed in battle in September, 1918. Received skin burns, of first degree, which healed in about six weeks. Also vomited a few times after being gassed. There was aphonia for six weeks. Kept in bed for about five weeks because of palpitation of the heart. Cough, sputum, and fever for a few days. Remained in the hospital nine weeks in all.

Patient returned to duty for two months. In February, 1919, on awakening one morning, he noted a sharp pain in the right side. He was sent to a base hospital for four

weeks, three of which were spent in bed. Fever, with temperature of  $103^{\circ}$  to  $104^{\circ}$ , was present for three to four days. "Nearly had pneumonia." There was a tendency to palpitation at the time of discharge from the base hospital.

**Present Illness.**—Since his return from France and return to civil life two months ago, the patient has been troubled somewhat by palpitation, particularly on retiring at night. He has tried a little hill-climbing in the past two weeks and noted moderate amount of palpitation. Four days ago there was a slight return of distress in the right side (axilla); no cough or other symptoms. Otherwise feels in good health.

**Physical Examination.**—Slight pyorrhea. Lungs normal to percussion and auscultation.

**Heart.**—Measures 3 by 9.5 centimetres, about one centimetre outside nipple line. Action regular, rate eighty-two. First sound at apex is snapping in quality; pulmonic second sound not accentuated; no murmurs. No tremor of fingers.

**Diagnosis.**—Effort syndrome.

**Discussion.**—This case is similar to many others found in the army. The signs and symptoms, essentially those of a lack of physical fitness, and the history of being gassed, and of "nearly having pneumonia" two months before I examined him, offer a sufficient reason for the condition termed effort syndrome. The history contains some details by which one may judge as to the probable severity of the gassing and of the respiratory affection. In the absence of other findings of abnormality, I do not put much weight on the left border of cardiac dulness being one centimetre outside the nipple line.

The pain in the right side may be due to pleural adhesions resulting from the chest infection. The possibility of this and the nature of effort syndrome was explained to the patient. He was advised as to regular exercise to regain his physical fitness. He rapidly returned to good health.

**CASE 20.—Housewife, aged thirty-two years. Referred with the diagnosis: "Myocarditis."**

**Past History.**—Influenza three years ago. Attacks similar to the present when in high school, and three years ago when last baby was born.

**Present Illness.**—Palpitation and faintness during the past two years. The duration is about ten minutes, and the cessation is gradual. Sometimes has the sensation as if the heart turned over. One flight of stairs causes no shortness of breath, if taken at an ordinary pace. Occasionally has pains, mostly occurring at the times of the menses. The attacks or symptoms are usually associated with nervous excitement. Feels she has had much trouble and reason to be nervous.

**Physical Examination.**—Fairly well developed, somewhat thin (weight one hundred and seven pounds).

**Heart.**—Impulse palpable in fifth space in left mid-clavicular line; no enlargement to percussion. At the apex there is a faint systolic murmur with the first sound; no diastolic murmur appreciated. Pulmonic second sound is louder than aortic second but is not accentuated. Considerable respiratory arrhythmia; heart rate about eighty-two.

**Social History.**—Children: Frederick, eleven years, six months; Eleanor, eight years, eight months; Pearl, two

years, six months. Both little girls appear lovable and fairly healthy; normal births. Birth of the boy was normal, but mother was under quite a nervous strain during pregnancy. Husband developed peculiarities shortly after she was married; was abusive to her during the first pregnancy. He continued this abuse through ensuing years and showed terrific temper, especially toward the boy, whom he frequently struck. Husband was committed to Psychopathic Hospital in 1916, sent to Westboro, and discharged in 1917. He came home and resumed his abuse toward his wife and son. Mother tried to have him arrested two or three times but changed her mind. The boy became cowardly, depressed, and prematurely old, while the mother became very nervous and easily upset.

In July, 1920, a visitor from the Boston Children's Aid Society went to the home and found the mother ill on a couch from the effects of the husband's brutality. She said he had knocked her down, kicked and stepped on her with heavy shoes, causing severe bruises; eye-glasses were broken and she was unable to get about. The husband was arrested and sent to the Cambridge Jail, where he was bailed out by his employer. He was later found guilty and placed on probation. In December, 1920, the court case was placed on file.

The patient had been examined by Dr. D— (an outside physician), who stated that she is a delicate, nervous woman, hypochondriacal and anæmic. He had treated her principally for pelvic irritation, due to the fact that she had not received proper care after the birth of her child.

Our contact with the patient has been one of fairly good coöperation, but we have felt that she never had a

proper environment in which to get well. Mr. S— was arrested again in September, 1921, was found guilty of non-support of his wife and children, and of being armed with a dangerous weapon. He was given eight months' sentence and was committed to the House of Correction. On November 1, 1921, the patient was operated on for complete hysterectomy and has gotten along well, but is worried about the approaching return of her husband from jail.

(The above lengthy social history may seem irrelevant to the subject of cardiology, but is quoted with the object of illustrating the futility of treating such cases from a purely medical standpoint.)

*Diagnosis.—Irritable heart.*

*Discussion.—*When this patient was first seen before the social history was obtained, my note on the record reads: "No evidence of true heart disease. Proper term is probably 'irritable heart' (*i.e.*, a normal heart in a nervous person). Environmental troubles could cause all these symptoms." It was hardly expected, however, that such a chapter of troubles would be disclosed. Can it be wondered that the patient was upset nervously? It would seem that the diagnosis obtains much support.

It has been my experience, when making the diagnosis of so-called irritable heart, to so frequently find in the social history data which might reasonably have given rise to the symptoms, that in all such cases an exploration of this part of the history is suggested. The above case well illustrates the value of so doing. Such patients are often sent to the heart specialist as probable cases of heart disease.

The diagnosis, "Myocarditis," is unjustifiable; there is

nothing in either the history or the examination to indicate real heart disease. The term "myocarditis" was probably employed, as is not infrequently true, because the physician thought there was something wrong with the heart, but could not find sufficient data on which to base the diagnosis of a valvular lesion. A diagnosis of this sort leads to nothing helpful and may even cause the overlooking of facts such as those brought out in the social history. It would be preferable to label such a case, "Probable heart disease, type undetermined," as this would stimulate further effort toward diagnosis.

### MISCELLANEOUS CONDITIONS

**CASE 21.**—Banker, aged fifty-four years.

**Complaint.**—Shortness of breath if walks rapidly, and some pain in left arm.

**Family History.**—Wife and one daughter in good health. No children dead; wife has had no miscarriages.

**Past History.**—Jaundice twenty-five years ago; complete recovery in a few weeks. At the age of twelve patient had a short attack of asthma. Denies other diseases when named in order. Four years ago, there was an attack of pain in the right flank; diagnosis that of renal stone. Advised to discontinue coffee and meat and has felt better. Now uses a little meat. Formerly was a hearty eater, including much meat.

The patient has been under considerable business strain. Five months ago he was all used up from over-work; felt better after several days' rest. Inclined to active exercise. He has never smoked, and has taken almost no alcohol. There has been a gain of ten pounds' weight in the past year.

*Present Illness.*—While climbing a mountain last summer patient noted a pain along the inner aspect of his left arm. Soon better and was able to continue the climb. Seven weeks ago he played four sets of doubles at tennis without apparent trouble.

During the past six weeks, however, when walking rapidly, there has been a distress under the head of the sternum. The sensation is described as "like that one gets after running too much." It is often associated with a similar distress over the left pectoral muscles and down the inner aspect of the left arm to the elbow. During the attack, which persists two to five minutes, he stops ostensibly to look in a store window. States that these attacks are so frequent that he has ceased taking the train to town, and goes by trolley so as to avoid meeting his numerous business friends and to be able to walk the few blocks to his office alone and at his own pace. Has noted, however, that sometimes if he is walking with some one and engaged in conversation, he may escape an attack.

Patient states that he has several times recently been told by other physicians that he was all right, and comes expecting he would be given a clean bill of health. (Much of the data listed above was obtained only by spending considerable time in taking the history, although the latter was not given unwillingly.)

*Physical Examination.*—Very well-developed man, not overweight. Pupils equal and react to light and distance. Teeth in good condition. No general glandular enlargement.

*Heart.*—Supraventricular dulness two by one and one half centimetres. No visible or palpable pulsation in the supra-

sternal notch. Impulse barely palpable one finger's breadth outside the nipple in the fifth and sixth spaces. Action regular, no murmurs, aortic second sound slightly accentuated. Heart rate seventy-two. Blood pressure; systolic one hundred and sixty, diastolic one hundred and ten. No suggestion of alternation noted (in taking blood pressure).

Urine clear, slightly acid, 1024, albumen and sugar absent.

Röntgen examination. Heart appears normal in size and contour, though there is a doubtful suggestion of enlargement of the left ventricle. The aorta appears elongated with a marked prominence of the knob to the left. Fluoroscopic examination not made.

*Diagnosis.*—Probably hypertensive heart disease. Definite angina pectoris. Some cardiac enlargement probable.

*Discussion.*—The above is clearly a case of angina pectoris. This diagnosis is based almost entirely on the history, which is consequently given somewhat in detail. The story of the patient's endeavor to walk alone, so as to be able to take his own pace, is quite impressive in the case of a man who has until recently played tennis with pleasure.

An etiological diagnosis of hypertensive heart disease is suggested by the blood-pressure record and the probable slight enlargement of the heart. Of course, before the diagnosis of hypertension can be definitely accepted, it must be shown that the blood-pressure levels are constantly elevated. Also a Wassermann test should be made and perhaps antisyphilitic treatment instituted. The presence of

cardiovascular syphilis must be considered because of the angina pectoris, but not, however, because of the hypertension.

This patient was seen but once prior to his going abroad. The presence and nature of angina pectoris was explained to him and, together with other advice, the need of additional examination was pointed out and apparently accepted. This man's history is cited partly as an illustration of the uncertainty as to the etiological type of heart present in some of our cases and the need of carefully watching such cases in order that disease may be recognized in its earliest phases.

**CASE 22.—Widow, twenty-one years of age.**

**Complaint.**—Pain over heart. Is said to have angina pectoris.

**Past History.**—The patient recalls practically no diseases. She was subject to weakness and fainting attacks during her childhood. At the age of fifteen the patient was married to a much older man; the marriage was arranged by her mother for financial reasons. There developed soon after a condition described as "nerves." Often the patient fainted from worry. She lived but eight months with her husband, who was later killed in an accident, and then for two years the patient was free from "weak spells." She has been a dancer and living a life of social excess. Denies venereal disease.

**Present Illness.**—About eight months ago, at night after a dance, a twinge was felt round the heart and the patient fainted. The next morning there were eighteen such attacks of a duration of two to three minutes. Two weeks later twelve similar attacks occurred while the pa-

tient was sitting in a chair. She did not definitely faint each time. With the first attacks the patient would stiffen. "As long as the pain keeps down I can get the better of it, but when it comes up (put hand over second left costal cartilage) I can't get my breath." The pain is described as gripping or squeezing; it does not radiate to arms, shoulders, or back. Ascending stairs causes palpitation for about ten minutes. A sensation of fatigue is present in the morning; the patient feels fine at times and down in the dumps shortly after.

For the past three weeks the patient has been kept in bed, but is not improving. Attacks of pain recur at irregular intervals, never following exertion.

*Physical Examination.*—Young woman, well developed. Pupils equal and react to light. Teeth in excellent condition. Tonsils appear normal. No general glandular enlargement.

*Heart.*—Impulse in fifth space inside left midclavicular line. No increased supraventricular dulness, percussion outline of the heart is within normal limits. Sounds clear, no murmurs, regular rhythm.

Reflexes normal. No skin lesions, thickening of tibiæ, or other signs to suggest syphilis.

*Further History.*—The friend at whose home the patient is visiting states that at the time of the attacks, the patient runs screaming round the house and will hurt herself unless prevented. At times it takes several persons to hold her in bed.

Family physician reports that the patient is in love with a young man of her own age, but is probably to marry an older man who has more money.

Wassermann test negative.

Röntgen examination shows a heart and aorta of normal contour and size.

*Diagnosis.*—Not true angina pectoris, or true heart disease.

*Discussion.*—At the age of twenty-one angina pectoris is of very rare occurrence. In the above case history, it should be noted that the attacks are not associated with exertion, but with emotional stimuli. The picture is that of a functional nervous condition, for which ample causation is found in the patient's history. The negative Wassermann reaction and normal Röntgen findings are of confirmatory value in excluding true angina. The screaming and activity of the patient during the attacks should immediately suggest that some other condition than true angina pectoris is present. This detail of the history would probably not have been obtained if a description of the attacks had not been sought from others than the patient.

The reason why this was not a real disease of the heart, but rather a nervous condition resulting from her numerous troubles, was explained in some detail to the patient, who appeared ready to listen carefully. The desirability of leading a more normal life was pointed out. Her physician reported that the attacks ceased and there was a rapid return of the patient's usual strength.

The above case forms a good contrast to Case 21.

**CASE 23.**—Housewife, forty-six years of age. Entered hospital for treatment of erysipelas of the face, chest, and arms.

*Past History.*—Measles when child; otherwise has enjoyed good health.

*Present Illness.*—Erysipelas started on the face and later extended to the chest and arms. Fever, varying from 101° to 104°, continued about sixteen days. Patient became much debilitated.

Three weeks later, pain in the joints and nodules in the superficial tissues of the limbs appeared; a diagnosis of erythema nodosum was made.

Toward the end of the eighth week (when the writer first saw this patient), the heart rate was showing marked variations, from normal to one hundred and fifty per minute. The periods of tachycardia occur several times per day and persist for several hours. (Three weeks ago a similar condition of regular tachycardia was present at intervals for about ten days.)

*Physical Examination.*—Fairly well developed and nourished. Moderate cyanosis of face, lips, ears, and fingertips; patient looks uneasy and complains of a pain in the right epigastrium. Cervical veins engorged and pulsating rapidly.

*Heart.*—Impulse barely palpable in fifth space at left midclavicular line. Borders of deep cardiac dulness within normal limits. Action regular, rate one hundred and fifty-six per minute; sounds tick-tack in quality, no murmurs appreciated.

Pulses barely palpable, regular, rate one hundred and fifty-six.

The liver extends from the sixth rib to three fingers' breadth below the costal margin in the mammillary line. The lower edge is distinctly tender on palpation. There is no edema of the extremities.

Digital pressure on the right vagus nerve in the neck, combined with the holding of a deep breath by the patient, slowed the heart rate markedly for a few seconds. On the sixth attempt, the heart rate suddenly dropped to eighty-two and remained at about that level. This was promptly followed by a radial pulse of normal rate and volume, disappearance of the engorgement of the cervical veins and the cyanosis of the face, and very evident relief to the patient.

The electrocardiogram, taken just previous to the use of pressure on the vagus, showed paroxysmal tachycardia of auricular origin. The tracing is reproduced in Fig. 20.

*Diagnosis.*—Paroxysmal tachycardia.

*Discussion.*—The successful abolition of the paroxysm required repetition of the methods used. There was evidently a considerable degree of circulatory failure, all of which, including the enlargement of the liver, disappeared after the return of normal rhythm.

The etiologic diagnosis is uncertain. The occurrence of the infections noted in the history offers a sufficient cause for this functional disturbance of the heart. Whether the latter was affected by toxins or was directly infected could best be determined at a future date. The paroxysms ceased and there was no further evidence of cardiac disturbance. It would be a good plan to examine such a patient after the lapse of a few months.

**CASE 24.**—Boy, aged four and one half years.

*Complaint.*—Very slow heart rate, and the possibility of convulsions.

*Present Illness.*—There is a history of convulsions which occur in the morning and last for a few minutes,

after which the patient appears normal. No other symptoms. He appears to be well and strong. Brought to a physician for treatment of the convulsions. On examination the heart rate was found to be about forty per minute, but on exercise the rate promptly doubled. Exercise is well tolerated. The physician suspected A-V heart-block and referred the patient for electrocardiographic examination.

*Family History.*—Contains no pertinent data.

*Past History.*—Patient had a normal birth and was breast-fed. There have been no previous illnesses.

*Physical Examination.*—Well developed and nourished. Reflexes normal. Teeth, tonsils, and glands normal.

*Heart.*—The impulse is in the fifth space just inside the nipple line; the borders of dulness are within normal limits. Sounds clear, no murmurs appreciated. When the patient is sitting quiet the heart rate is about forty-four per minute, but the rate promptly becomes about eighty if he stands up or exercises. The action is essentially regular save for the change of rate. There are no visible waves in the cervical veins during the long diastoles of the heart. The pulses are of good volume and tension; there is no pulse deficit.

The electrocardiogram shows sino-auricular block. At the slow rate the rhythm originates in the auriculo-ventricular node, and in the sinus node at the faster rate, *i.e.*, there is a shifting of the pacemaker. The P-R interval measures 0.14 second.

*Diagnosis.*—Sino-auricular block.

*Discussion.*—This is an example of a functional condi-

tion of the heart of unknown origin, and, in the absence of other evidence of abnormality in the heart, should not be interpreted as proof of disease. It must in no sense be confused with true heart-block, *i.e.*, between the auricles and ventricles, an error that was made by his physician after receiving the report of the electrocardiogram.

The entire lack of the symptoms and signs of heart failure in this patient should suggest an essentially normal heart. The approximate doubling and later halving of the heart rate is a well-known sign of sino-auricular block. The normal appearance of the venous pulse is helpful to those accustomed to using this finding.

There is no doubt that, in a case such as the above, the electrocardiogram is of distinct value in establishing the diagnosis. It may be noted that the nature of the convulsions has not been discussed. Observation in a hospital for a period of ten days failed to show anything abnormal.

**CASE 25.**—Married woman, aged twenty-four years, born in the West Indies.

**Complaint.**—Palpitation and fainting.

**Present Illness.**—Patient is referred from the Maternity Clinic of the Boston Homeopathic Hospital.

There has been a tendency to a rapid heart rate (patient indicates by motion of the hand a rate estimated to be about one hundred) and fainting. The pregnancy is now in the fourth month; there has been much morning sickness with vomiting, but recently this has ceased.

**Past History.**—Measles, whooping cough, and an occasional mild sore throat are the only diseases recalled. Otherwise the patient has enjoyed good health; could run and play well.

**Physical Examination.**—Well developed but thin.

**Heart.**—The impulse is palpable in the fifth space inside the left midclavicular line; percussion borders within normal limits. There is a soft systolic murmur with the first sound at the apex; the sounds are otherwise normal. Some variation in rate with respiration is present.

**Electrocardiogram.** During the taking of this the patient fainted. She was placed in a horizontal position on a couch, and recovered in a few minutes. The record showed a normal sinus rhythm of a rate of about eighty-eight, before and after the attack; during the few minutes' loss of consciousness A-V rhythm, rate sixty-nine, was present.

**Diagnosis.**—Sino-auricular block interrupting normal rhythm.

**Discussion.**—The patient was probably suffering from an irritable heart induced by the recent period of severe nausea and vomiting of pregnancy. The shifting of the pacemaker from the sinus to the A-V node was most likely due to the action of the vagus nerve. While the patient was unconscious the heart sounds were regular and of good quality; the slowing of the rate led to the conclusion that atrio-ventricular rhythm was present.

Neither in the history nor in the electrocardiogram of this patient is there evidence of actual disease of the heart. It was predicted that the patient would be in good health as soon as the effects of her recent illness had disappeared.

**CASE 26.**—Hospital interne, twenty-eight years old.

**Complaint.**—Irregular heart action occurring during an acute nasal sinusitis.

**Past History.**—He has had measles when an infant, typhoid fever at the age of eleven, and tonsillitis of five days' duration about eight months ago. Otherwise has

enjoyed excellent health; has never noted any signs or symptoms of cardiac disturbance.

*Present Illness.*—Four days ago there was a sudden chill followed by fever and an aching all over the body. Other symptoms are,—loss of appetite, moderate nausea, and a single attack of vomiting twenty-four hours ago. There has been a temperature ranging from 101° to 102° for four days.

Yesterday tenderness developed over the right antrum, and there is a feeling of fulness in the right cheek and forehead. Coryza was present two days before the chill, but has become much less since then. Marked irregularity of the heart has been present during the past twenty-four hours.

*Physical Examination.*—Patient looks prostrated. There is a slight nasal discharge of a purulent type, mostly from the right nostril.

*Heart.*—Impulse in the fifth space just inside the nipple line. The right border of deep cardiac dulness is two centimetres and the left border nine centimetres from the midsternum. There are no murmurs. The rhythm is a regular irregularity; coupling is often heard, and at times there are three or more regular beats interrupted by premature sounds. During the coupling, alternate large and small waves are readily distinguished by palpation of the pulse. Inspection of the venous pulse in the neck shows the auricular wave preceding the waves that are simultaneous with the beat following the longer pauses.

The arteriogram shows premature beats alternating with normal beats. The tracing is reproduced in Figure 5.

The electrocardiogram shows ventricular premature beats interrupting a sinus rhythm.

The antrum was drained and in two days the irregularity of the heart disappeared. Convalescence was uneventful.

*Diagnosis.*—Acute sinusitis, ventricular premature beats.

*Discussion.*—The picture is that of an acute infection of considerable severity. The appearance of extrasystoles is not uncommon in such cases. The condition is to be sharply distinguished from true pulsus alternans. A careful noting of the time of the beats, particularly on auscultation, made it clear that some of the beats were premature; such would be the usual explanation of the coupling. Sino-auricular block, or true heart-block, with a dropping of every third beat, would be a possibility, but could be excluded by the occasional runs of normal beats, which gave a gauge as to the usual interval that should separate the beats if all were of normal origin. Also the waves in the radial pulse showed a marked and regular alternation in volume, which is not a feature of dropped beats. By means of the data discussed in Chapter XVII it was easy to exclude sino-auricular block, or heart-block, and to confidently diagnose the extrasystoles. The graphic records were conclusive.

This heart was doubtless affected by the acute general toxemia, but since the recovery was prompt and complete, an actual infection of the heart can be confidently dismissed.

**CASE 27.**—Lawyer, aged forty-five.

*Complaint.*—Cyanosis and irregular action of the heart.

*Past History.*—Has always enjoyed good health. Could run as well as other boys, but was not so strong. Was told, when about nineteen years old, that his heart was irregular and has received many opinions from different

physicians; some said the condition was harmless, and others, serious. The doctors are always examining his heart; the condition is called functional, as a rule. Patient noted that his heart was regular if he had fever.

*Present Illness.*—During the past three years his friends have frequently commented upon the duskiness of his face and hands, though he himself was less conscious of any change in color. Has noted that after playing golf or being out in the air, the blueness of the skin is less prominent. Feels in excellent health, enjoys three sets of tennis, is now planning a fishing trip into the Maine woods. Referred for electrocardiogram.

*Physical Examination.*—Well developed and nourished. Face and hands have dusky slate-colored tinge. This does not show accentuation at the lips and ears, or fingertips. The "cyanosis" is absent above the wrists and below the level of the collar; it is especially marked at the base of the nails, at the lunulæ. The dusky hue is absent inside the mouth. On pressure the skin blanches, but a very definite bluish color remains.

*Heart.*—Apex impulse in fifth space inside nipple line, no evidence of enlargement disclosed by percussion. Split first sound at apex. Regular rhythm frequently interrupted by premature beats, rate seventy-two. One hundred hops well tolerated; rate accelerated to one hundred and twenty and became perfectly regular. As the heart slowed, the irregularity returned at the rate of ninety. No edema.

*Diagnosis.*—Premature beats, argyria.

*Further History.*—No history of the use of silver nitrate. Some digestive trouble about ten years ago for which he took a liquid medicine, of red color, for a few

months. Twelve years ago argyrol was prescribed for the treatment of a catarrhal condition of the nose. Patient liked the effect so well that, on his own initiative, he continued the use of this at least twice a day for eight years. Is satisfied that he swallowed considerable of the drug. The change in the color of the skin appeared gradually, four years ago.

The electrocardiogram showed premature beats of ventricular origin.

*Discussion.*—The duskiness of this man's face and hands was of the degree that is rarely present save in patients almost in extremis, and yet he admitted no impairment in health. In addition to this reason to question the genuineness of the cyanosis, a more careful examination showed, as described above, that it was abnormal in its distribution and its non-disappearance on pressure. It is noteworthy that the discoloration was limited to those parts of the body surface that are readily accessible to light; this patient stated that he rarely rolled up his sleeves or wore a bathing suit. In a sense the patient has become somewhat like a photographic plate. The most reasonable explanation of the lessening of the blueness of the skin after exercise in the open air is that the natural redness of the skin so induced tended to mask somewhat the underlying pigmentation.

Cases of argyria from the use of argyrol appear to be rare, but it is the probable offender in this case. Since the patient admitted the habit of swallowing the drug freely, it is the less surprising that argyria ensued.

The cardiac irregularity presented all the clinical features of extrasystoles. This history of an irregularity for years, with the continuance of normal health and vigor,

should suggest the arrhythmia to be of the simpler type. The rhythm became regular after acceleration of the rate by exercise; the patient had made a similar observation when fever was present, *i.e.*, when the heart rate was increased.

**CASE 28.**—Physician's daughter, aged seven years.

**Chief Complaint.**—Sent home by school physician with the report of heart disease.

**Family History.**—Parents and two sisters living and well.

**Past History.**—Whooping cough and chicken-pox one year ago. No rheumatic fever, growing pains, chorea, tonsillitis, or scarlet fever. Has always been strong; plays with girls of an older age; can run well. During the past two years has been kept out of school some because of being underweight. By means of a generous diet, more fresh air, and rest, she has gained weight in the past year.

**Physical Examination.**—Fairly well developed and nourished. Mucous membrane of good color. Tonsils moderately prominent, no evidence of disease. Teeth in excellent condition. A few small, non-tender cervical glands. Thoracic wall very curving (moderate pigeon-breast).

**Heart.**—Impulse in fifth space just outside of nipple line. Border of cardiac dulness appears within normal limits. Loud systolic murmur in second and third left interspaces near the sternum transmitted to right and into neck. This murmur is much diminished on full inspiration. Respiratory arrhythmia present.

Hemoglobin, ninety per cent.

**Diagnosis.**—No heart disease.

**Discussion.**—The only suggestion of an affection of

the heart is the murmur of maximum intensity at the pulmonic area. In the absence of other abnormal findings it is a safe dictum to disregard such murmurs. The causation of this type of murmur is uncertain, but, in my opinion, if anemia is absent, the most probable explanation is the relation of the conus arteriosus and great vessels to the underlying thoracic wall. (See Chapter XIV.) It was noted that this little girl's thorax was somewhat abnormal in shape.

**CASE 29.—Housewife, aged forty-two.**

**Family History.**—Unimportant.

**Past History.**—Never well and strong. Best weight one hundred and six pounds, now is ninety-five. Recalls none of the diseases of childhood.

Husband left her eight years ago. There are no children. Patient inclined to worry. Thought she had acquired syphilis from her husband, but there have been no symptoms in support of that diagnosis, and several Wassermann tests have been negative.

**Present Illness.**—Some backache when she has been on her feet long. Also notes a drawing sensation as though a weight on the anterior abdomen. Comes to Heart Clinic as a case of probable valvular heart disease. A soft systolic murmur has been found at the apex, and the patient is said to have shortness of breath and pain on exertion. Tires easily.

After careful questioning it is clear that the patient is not short of breath on exertion that is reasonable, in my opinion, for one of her sex, age, and physique. Also the pain is found to be that noted above, or rather, a drawing sensation in the epigastrium. This symptom is equally

liable to be present when the patient has simply been standing too long.

*Physical Examination.*—Poorly nourished woman with stature conspicuously that associated with visceroptosis. The chest is flat, the costal angle narrow, the lower abdominal wall sagging, and there is a prominent anterior curve to the lumbar spine.

*Heart.*—The impulse is palpable in the fifth space at the midclavicular line; the percussion borders of the heart are within normal limits. Action regular, no definite murmurs appreciated. The first sound at the apex is somewhat crescendo in type.

The pulses are of fair volume and tension. Blood pressure 130/85. Reflexes lively.

*Diagnosis.*—Not true heart disease. Visceroptosis.

*Discussion.*—The soft systolic murmur at the apex is probably what I have described as a crescendo type of first sound. It is known that in some normal hearts the first sound appears to begin with a soft murmur, or has a crescendo quality which closely simulates the type of first heart sound often present in cases of mitral stenosis. An apical systolic murmur, without other abnormal findings, is not evidence of disease of the heart.

It is proverbially difficult to estimate slight degrees of diminished tolerance to exercise. As said in a previous chapter, it is not merely the production of symptoms on exertion, but of symptoms *that are untoward* for the particular patient under examination. Experience is probably the best guide in forming one's judgment. The symptom of pain on exertion in this case became largely valueless as an indication of cardiac insufficiency, when on

further investigation it was found to be a dragging sensation in the abdominal wall.

This patient's physical development, or lack of it, offers a sufficient cause for her symptoms. The exaggerated curve of the spine and the pendulous abdominal wall are well-known physical handicaps. Her marital troubles would not conduce to the possession of good health.

The above case also serves as an illustration of the precept that the examiner must keep in mind the patient as a whole and not be too ready to blame the heart. Sir William Osler is often quoted as saying that he who thoroughly knows syphilis knows all medicine; with almost the same justification it may be stated that he who knows all cardiology must be familiar with all medicine.



## CASE INDEX\*

- Aneurism, 9  
Angina pectoris, 10, 12, 21  
    simulation of, 22  
Aortic regurgitation, 4, 7, 8  
Argyria, 27  
Arteriosclerotic heart disease, 11, 12  
Asthma, 7  
Atrio-ventricular rhythm, 25  
Auricular fibrillation, 3, 5, 8, 14, 15, 16  
Autopsy, 6, 9, 17  
Blood culture positive, 6  
Capillary pulse, 4, 7  
Chest deformed, 14, 28  
Chill, 5, 6, 26  
Congenital heart disease, 17, 18  
    coarctation of aorta, 17  
    dextrocardia, 18  
    patent ductus arteriosus, 17  
    patent foramen ovale, 17  
    patent interventricular septum, 17  
Corrigan pulse, 4, 7, 8, 9  
Cough, 3, 9, 15  
Cyanosis, 5, 7, 9, 14, 16, 23, 27  
Death, sudden, 14  
Diphtheria, 16  
Doubling of rate, 24  
Duroziez's sign, 4  
Dysphagia, 9  
Dyspnœa (see shortness of breath)  
Edema, 5, 7, 9, 16  
    slight, 4, 13, 15  
Effort syndrome, 19  
Electrocardiogram, auricular hypertrophy, 3  
Emaciation, 5, 16  
Embolism, 6, 14  
Exercise tolerance, 29  
Extrasystoles, 3, 16, 26, 27
- Family history, positive findings, 2  
Fever, 5, 6, 26  
Gallop rhythm, 16  
Gonorrhœal arthritis, 7  
Heart-block, 9  
Heart failure, of definite degree, 2,  
    3, 5, 7, 8, 9, 14, 15, 23  
Hemoptysis, 3  
Hypertensive heart disease, 13, 14, 21  
Hyperthyroidism, 15  
Impulse, diastolic, 2, 4  
    diffuse 2, 4  
    outside nipple line, 4, 5, 7, 8, 9, 13,  
        14, 15, 19, 21  
    systolic retraction of, 2, 4  
Irritable heart, 20, 25  
Liver, enlarged, 9, 23  
Mitral involvement, 1  
    regurgitation, 1, 2, 3, 4  
    stenosis, 2, 3, 4, 5  
Murmurs, absent, 6, 17, 18, 19, 21, 22,  
    23, 24, 26, 27  
    crescendo at apex, 5, 29  
    diastolic at apex, 2, 3, 4, 5  
    diastolic at base, 4, 7, 8, 9  
    "presystolic," 5, 29  
    pulmonic, 28  
    systolic at apex, 1, 2, 3, 4, 7, 8, 9,  
        11, 13, 14, 15, 16, 20, 25, 29  
    systolic at base, 4, 7, 9, 10, 11, 12, 28  
"Myocarditis," 20  
Nephritis, 13  
Orthopnoea 2, 7, 14  
Pain, arm, 10, 12, 21  
    jaw, 10  
    neck, 10, 12  
    precordia, 2, 10, 22  
    shoulder, 2, 21  
    side, 5, 6  
    sternal, 10, 12, 21

\* The numbers refer to cases and not to pages.

- Palpitation, 5, 11, 15, 19, 20, 22, 25  
Paroxysmal tachycardia, 23  
Pericardial adhesions, 4  
    friction, 2  
Pericarditis, acute, 2, 6.  
Pistol-shot in artery, 4  
Premature beats, 3, 16, 26, 27  
Pulmonic murmur, 28  
    second sound accentuated, 2, 5  
Pulsation, below left scapula, 2, 4  
    suprasternal, 9, 12  
Pulse, capillary, 4, 7.  
    Corrigan, 4, 7, 8, 9  
    deficit, 3, 14  
    pseudo-alternation, 26  
Rheumatic heart disease, 1, 2, 3, 4, 5  
Rhythm, atrio-ventricular, 25  
    coupled, 26  
    gallop, 16  
Röntgen findings, positive, 1, 8, 9, 10,  
    12, 18, 21  
Septic heart disease, 5, 6  
Shortness of breath, 2, 3, 4, 7, 8, 9, 14,  
    15, 23, 29  
Sino-auricular block, 24, 25  
Social history, 20  
Supraventricular dulness, 9, 10, 12  
Symptoms, absent, 1, 18, 24  
Syphilis, cardiovascular, 7, 8, 9, 10  
Thrill, at apex, 5, 11  
Traube's double sound, 4  
Tricuspid lesion, 6  
Urine, red blood cells in, 6  
Visceroptosis, 1, 29  
Vomiting, 5, 16, 26  
Weakness, 5, 13.

# INDEX

- "A" wave, 51, 56, 57, 62, 211, 215  
Aberration of the ventricular waves, 76, 77, 80  
Abortive beats (see pulse deficit)  
Abscissæ, polygraph, 54  
  electrocardiogram, 70  
A-C interval, 57, 62  
Aconitine, 197, 208, 213  
Activity of heart disease, 99, 102  
Adams-Stokes' syndrome, 216, 256  
Adrenalin, 208, 213, 266  
  test, 162  
Alcohol, 127, 267  
Allbutt, C. A., 126, 127, 142, 151, 186, 187, 190  
Allen, F. M., 150, 153, 157  
Allen, H. B., 103  
Ammonia, 267  
Amyl nitrite, 41, 192, 267  
Anaphylaxis, 213  
Anemia, 123, 152  
Aneurism, 46, 123, 129, 241  
  cardiac, 128, 244  
  frequency, 137  
  murmurs in, 133  
  non-syphilitic, 130, 135  
  ventricular, 128  
Angina pectoris, 45, 128, 137, 147, 152, 155, 184  
  diagnosis, 189  
  etiology, 184  
  mock, 190, 191  
  predisposing causes, 193  
  prognosis, 192  
  symptoms, 187  
  treatment, 192  
Anthrax, 135  
Aorta, 22  
  calcification in, 129, 136  
  coarctation, 174  
  dilated, 35, 38, 47  
  elongated, 35  
  isthmus, 174  
  Röntgen findings, 133, 134  
Aortic insufficiency, 32, 34, 39, 40, 111, 113, 115, 128, 134, 136, 226, 237  
  functional, 100, 229  
Aortic stenosis, 111, 113, 128, 174, 230  
Aortic vestibule, 30  
Aortitis, 185, 186  
  non-syphilitic, 135  
  syphilitic (see cardiovascular syphilis)  
Apex, movement of, 24, 85  
  impulse, 32, 33, 85, 152  
  fixed, 112  
  in mitral stenosis, 104  
  retraction of, 104  
Aphasia, 122  
Aphonia, 110, 181, 173  
Apocynum, 265  
Arrhythmia, 92, 195  
  absolute, 210  
  combined, 83, 220  
  effect of exercise on, 210  
  respiratory (see sinus arrhythmia)  
Arsphenamine, 124, 138, 139, 141  
Arterial signs, 227, 228, 229  
Arteries, aorta (see)  
  to bundle branches, 19  
  carotid, visible pulsation, 32  
  coronary, 19, 20, 82  
    occlusion, 77  
    sclerosis, 128, 185  
    spasm, 185  
    thrombosis, 190  
  enlarged on back of abdominal cavity, 174  
  internal mammary, 21, 239  
  "pipe-stem," 145  
  pulmonary, 21

## INDEX

- Arteriosclerosis, 135, 185, 208  
 Arteriosclerotic heart disease, 142  
     blood pressure in, 145  
     diagnosis, 146  
     etiology, 142  
     pathology, 143  
     prognosis, 147  
     symptoms and signs, 144  
     treatment, 147  
 Aschoff bodies, 98  
 Asphyxia, 213  
 Atropine, 196, 216, 218  
 Auricle, left, 16  
     enlargement of, 38  
     right, 15, 37  
         enlargement of, 32  
 Auricular appendix, left, 16  
     right, 16  
 Auricular fibrillation (see fibrillation)  
 Auricular flutter (see flutter)  
 Auricular systole, 23  
 Auriculo-ventricular bundle, 18  
     branches, 19  
     rhythm, 57, 75  
 Auscultation, 39, 40  
     direct, 40  
  
 "B" wave, 56, 215  
 Bainbridge, F. A., 28  
 Bamberger's sign, 238  
 Barium chloride, 197, 208, 267  
 Baths, 158, 252  
 Bayliss and Starling, 66  
 Benda, 126  
 Bennert, R., 135  
 Benzyl benzoate, 159  
 Billings, 124  
 Bleeding (see venesection)  
 Block, arborization, 83  
     auriculo-ventricular (see heart-block)  
     intraventricular, 82, 83  
     sino-auricular, 58, 75, 196  
 Blood, chemistry, 153  
     cultures, 123, 124  
     flow, velocity of, 230  
     white count, 123  
 Blood pressure, 90, 219  
     in aortic insufficiency, 42  
     in cardiovascular syphilis, 133  
     determination, 42, 43  
     effect of digitalis, 257  
     elevated, 152  
     fall, 190  
     femoral, 43, 90, 228  
     hydremia, 254  
     instruments, 42  
         pulse pressure, 90  
 Books, 158  
 Bowels, 157, 252  
 Bowle, 39  
 Broadbent's sign, 112  
 Broken compensation (see heart failure)  
 Bromides, 200, 204  
 Brooks, H., 128  
 Brugsch and Schittenhelm, 38  
 Bundle of His (see auriculo-ventricular bundle)  
 Burton-Opitz, R., 107  
  
 "C" wave, 54  
 Cacodylate of soda, 124  
 Cactin, 267  
 Caffein, 266  
 Caffein sodiobenzoate, 268  
 Calcification of aorta, 136  
 Camphor, 267  
 Capillary electrometer, 66  
 Carcinoma of heart, 238  
 "Cardiac neurosis" (see effort syndrome)  
 Cardio-thoracic ratio, 46, 90  
 Cat unit (see digitalis)  
 Cautley, E. 172  
 Cerebral hemorrhage, 151  
 Chill, 120, 121  
 Chloroform, 164, 197, 208, 266

- Chordæ tendineæ, 17, 98  
Chorea, 31, 97, 119  
Christian, H. A., 259  
Circus movement, 201, 205, 209, 268  
Classification of heart disease, 91  
    New York, 93  
Claudication, 186  
Climate, 253  
Clothing, 253  
Coffee, 198  
Congenital heart disease, 170  
    symptoms, 170  
    treatment, 174  
Constipation, 157  
Contour of heart, 36, 47  
Contraction, amplitude of, 28  
    law of, 26  
    wave, 25, 72  
Conus arteriosus, 16, 21, 38, 235  
Convallaria, 265  
Convulsions, 122, 170  
Cor bovinum, 227  
Coronary artery (see arteries)  
    sinus, 18, 20  
    valve, 20  
Corpus Aurantii, 17  
Cotton, T. F., 73, 286  
Cough, 31  
    brassy, 131  
Councilman, Mallory, and Pearce, 165  
Coupled rhythm (see pulse, bigeminal)  
Crampton, C. W., 44  
Curran, 19  
Cushney, Marris, and Silberberg, 257  
Cyanosis, 32, 170  
  
"D. A. H." (see effort syndrome)  
Da Costa, J. M., 175  
Danzer, C. S., 46  
Death, sense of impending, 187, 189  
    sudden, 155, 168, 187, 188  
Dextrocardia, 170  
Diabetes, 149, 153, 156  
Diastasis, 24  
Diastole, 24  
Dicrotic notch, 55, 56  
Diet, 252  
    during administration of mercury, 139, 140  
    in arteriosclerosis, 148  
    in hypertension, 156  
Digitalis, 117, 148, 169, 197, 204, 207,  
    208, 212, 257, 270  
    administration, 260  
    cat unit, 260  
    effect on electrocardiogram, 72, 258  
Eggleston method, 260  
intramuscularly, 265  
preparation, 258, 264  
standardization, 259  
    T wave in, 72, 258  
Dilatation of heart, 28, 236, 239, 254  
Diphtheria, 135, 213  
Diphtheria, the heart in, 165  
    diagnosis, 168  
    etiology, 165  
    pathology, 165  
    prognosis, 168  
    symptoms, 166  
    treatment, 169  
Diuretics, 155, 157, 268  
Diuretin, 155, 268  
Dizziness, 145, 179  
Dominant rhythm, 53, 59, 62  
Double shock, 35  
Dress, 253  
Dropped beat (see heart-block)  
Drury and Iliescu, 272  
Ductus arteriosus (Botalli), 29  
    patent, 171, 173  
Dulness, apex left lung, 106  
    cardiac, 37, 38, 85  
        deep, 38, 86  
        left border, 37, 38, 86, 105  
        right border, 37, 86, 105  
        superficial, 38, 86  
below scapula, 238

## INDEX

- supracardiac, 38, 87, 132, 145, 173  
beside thoracic spine, 132
- Duroziez's sign, 229
- Dyspnœa, 32  
treatment, 254
- Ectopia cordis, 171
- Edelmann, 66
- Edema, 33, 152, 155, 184, 252, 255
- Edwards, A. R., 98
- Effort syndrome: irritable heart, 175  
diagnosis, 177  
etiology, 175  
prognosis, 177  
symptoms and signs, 176  
treatment, 178
- Eggleson, C., 260
- Einthoven, W., 66, 67, 68  
triangle, 69
- Electrocardiogram, 154  
in dextrocardia, 171  
in mitral stenosis, 110  
normal, 69  
standardization, 68
- Electrocardiography, 64  
history, 65  
the instrument, 67  
value of, 64
- Electrodes, 67
- Embolism, 110, 122, 167, 271  
"crossed or paradoxical," 173  
septic, 120
- Embryology, 29
- Emphysema, 86
- Empyema, 119
- Endocarditis  
acute, 225  
bacterial (see septic heart disease)  
infectious (see septic heart disease)  
malignant (see septic heart disease)  
statistics, 98, 111  
ulcerative (see septic heart disease)
- Enlargement of heart, 112, 152, 227, 236
- of left ventricle, 32, 38  
of right heart, 32, 184  
of right ventricle, 25, 34, 38, 104
- Epigastric pulsation, 32, 104, 237
- Epinephrin (see adrenalin)
- Epistaxis, 152, 170
- Erysipelas, 135
- Erythrol tetranitrate, 194
- Eustachian valve, 29
- Ewart's sign, 238
- Ewing, E. M., 54
- Examination, methods of, 31
- Exercise, 41, 178, 188, 249  
effect on arrhythmias, 199, 200  
physiology of, 27, 218  
tolerance, 31, 43, 45, 93, 177, 183
- Extrasystole, 52, 53, 58, 146, 197  
auricular, 58, 59, 76  
differential diagnosis, 199  
etiology, 197  
interpolated, 58, 219  
nodal, 59, 76  
pause after, 59  
prognosis, 200  
symptoms and signs, 198  
treatment, 200  
ventricular, 58, 59, 77, 221
- Eye, fundus of, 153
- Facies, 104
- Fatigue, 31, 183
- Fever, irregular, 122, 130  
"FF" waves, 61
- Fibrillation, auricular, 54, 57, 61, 78, 146, 167, 208, 268  
diagnosis, 211  
etiology, 208  
flutter-fibrillation type, 79  
pathology, 210  
prognosis, 212  
symptoms and signs, 210  
treatment, 212
- Fibrillation, ventricular, 80, 187, 258, 266

- Fingers, clubbing of, 104, 170  
  painful areas in, 122
- Fleming and Kennedy, 166
- Flint, Austin, 39, 109
- Fluid intake, 157, 252, 255
- Flutter, auricular, 61, 62, 78, 202, 205,  
  268  
  diagnosis, 206  
  etiology, 205  
  impure, 79  
  prognosis, 207  
  symptoms and signs, 206  
  treatment, 207
- Focus of infection, 97, 114, 116, 175,  
  178, 247
- Foramen ovale, 29  
  closure of, 173  
  patent, 172
- Frey, W., 271
- Friction at base, 132  
  pericardial, 35, 238  
  pleuro-pericardial, 239
- Friedreich, 112
- Functional conditions, 183
- Fundus of eye, 153
- Gallop rhythm, 35, 167, 215
- Gangrene, 122
- Gannet, 40
- Gaskell, 27
- Gastric symptoms, 151, 255  
  treatment, 193
- Gonorrhea, 119, 135
- Goodpasture, E. W., 160, 161, 164
- Graham Steel murmur, 109
- Graphic methods, 49
- Gross, L., 82
- Gummata, 128
- "H" wave, 56, 215
- Hands, cyanosis of, 176  
  pain in, 122, 188
- Harding, M. E., 167
- Hart, T. S., 266
- Hatcher and Brody, 260
- Hay, J., 51
- Headache, from cerebral congestion,  
  144, 147
- Heart, anatomy, 15  
  low-lying, 32, 47  
  normal, 84  
  normal position, 15
- Heart-beat, cessation of, 214, 215  
  mechanism, 25
- Heart-block, 61, 75, 78, 80, 133, 134,  
  147, 166, 168, 199, 206, 213  
  complete, 62, 80  
  diagnosis, 216  
  etiology, 213  
  partial, 61, 80  
  pathology, 214  
  prognosis, 217  
  symptoms and signs, 214  
  treatment, 217
- Heart disease, active or inactive, 99,  
  102  
  in anemia, 92  
  beer heart, 92  
  classification, 91  
  in obesity, 92  
  potential, 113, 134  
  reliable signs of, 48  
  tumors, 92  
  types of (etiological), 91
- Heart failure, 31, 45, 92, 183, 192, 234  
  relation to infection, 100, 183
- Heat, 192
- Hellebore, 265
- Hemiplegia, 122, 271
- Hemopericardium, 240
- Hemoptysis, 106, 110
- Henderson, Y., 107
- Henderson and Barringer, 28
- Henderson and Johnson, 23
- History, 31, 84, 97, 136, 203  
  etiological diseases, 31
- Hoarseness, 110, 131
- Hoffman's anodyne, 193

- Hoover, C. F., 38  
 Hopkins, A. H., 150  
 Huchard's table, 190, 191  
 Hydremia of blood, 254  
 Hyperesthesia, 176, 177  
 Hypertension (see hypertensive heart disease)  
 Hypertension, effects of digitalis on, 258  
 Hypertensive heart disease, 136, 149, 219  
   diagnosis, 154  
   etiology, 149  
   pathology, 150  
   physical signs, 152  
   prognosis, 155  
   symptoms, 151  
   treatment, 156  
 Hyperthyroidism, 149, 161  
 Hyperthyroidism, the heart in, 160, 177, 208  
   diagnosis, 163  
   etiology, 160  
   pathology, 160  
   prognosis, 164  
   symptoms and physical signs, 161  
   treatment, 164  
 Hypertrophy, 236  
   ventricular, 73  
 Hypothyroidism, 72  
  
 Idio-ventricular rhythm, 81  
 Impulse, aortic, 35  
   cardiac (see apex)  
     absent, 239  
     character, 34  
     diastolic, 34  
     downward, 237  
     maximum, 33, 85  
     movement of, 33  
     out and downward, 34, 227, 237  
     position, 33, 34  
     retraction of, 34  
  
 Indigestion, 31  
   "acute," 188  
 Infarcts, 122  
   of myocardium, 77  
 Infection (see focus of)  
 Influenza, 121, 135, 185  
 Infundibulum (see conus arteriosus)  
 Inman, T. G., 145, 148  
 Insomnia, 255  
 Inspection, 32  
 Interventricular septum, 30  
   patent, 30, 171, 173  
 Iodide of potassium, 185, 140  
 Irregularity (see arrhythmias)  
 Ischemia (see myocardium)  
 Isometric period, 25  
  
 Jaundice, 104  
  
 Kidney, function of, 153, 154  
 Kölliker and Müller, 65  
 Krumbhaar, E. B., 162  
  
 Lancisi, 126  
 Lead, 142  
 Leads, 68  
 "Leaky valves," 251  
 Lesions, structural, 93  
 Levison, L. A., 125  
 Levy, A. G., 266  
 Lewis, T., 24, 45, 48, 50, 53, 56, 61, 64, 67, 68, 72, 81, 176, 178, 197, 200, 202, 205, 206, 207, 208, 209, 213, 214, 217, 236  
 Lewis, Drury, and Iliescu, 272  
 Lewis, Meakins, and White, 81  
 Lewis, Feil, and Stroud, 205, 207, 209  
 Ligamentum arteriosum, 21  
 Litten's sign, 112  
 Liver, pulsation, 234  
   swelling of, 184, 202, 234  
 Ludwig, angle of, 33  
  
 MacCallum, W. G., 17, 143

- Mackenzie, J., 49, 97, 112, 187, 225, 234, 235  
Magnus-Alsleben, 166  
Malaria, 135  
Massage, 250  
of heart, 256  
McCrae, 98  
Meakins, J. C., 81  
Meakins and Gunson, 178  
Means, J. H., 162  
Measles, 97, 135  
Mediastinal tumors, 137  
Meningitis, purulent, 121  
Mercury, 135, 139, 141  
Metabolism, basal, 154, 162  
Mines, G. A., 206  
Mitral insufficiency, 102, 115, 231  
relative, 132, 231, 232  
Mitral stenosis, 34, 38, 39, 88, 99, 103, 115, 136, 208  
facies, 104  
murmurs, 107  
Monckeberg's sclerosis, 143  
Morphine, 193, 213  
Moschcowitz, E., 151  
Müller, F., 142, 163  
Murmurs, 40, 41, 121, 225  
apical, 41, 89  
Austin Flint, 132  
basal, 41, 89  
crescendo, 88, 108  
diastolic, 39  
at base, 100, 226  
double, 172  
experimentally produced, 172  
Graham Steel, 109  
mid-diastolic, 233  
in mitral stenosis, 107  
pitch, 39, 40  
presystolic, 107, 108  
production of, 106  
systolic, at apex, 103, 132, 146, 153, 176  
at aortic area, 132  
at pulmonary area, 89, 172, 234  
timing of, 34  
tricuspid, 233  
Muscarine, 197, 213  
Musser, J. H., Sr., 192  
Myocarditis, 235  
toxic, 160, 165  
Myocardium, 97, 98, 115, 127, 212, 235  
anatomy, 17  
compensatory circulation, 185  
degenerative changes, 144  
in hypertension, 151  
in hyperthyroidism, 160  
ischemia, 185, 186, 191  
in mitral stenosis, 103  
Myogenic theory, 26  
Nephritis, 122, 150, 153, 156, 238  
Nerves, 20, 26  
phrenic, 21  
recurrent laryngeal, 22, 110, 131, 173  
sympathetic, 21, 26, 243  
vagus, 21, 26, 29, 195, 196, 203, 213, 216, 217, 257, 267, 269  
"Nervous heart" (see effort syndrome)  
Nervousness, 204  
Neuro-circulatory asthenia (see effort syndrome)  
Nicotine, 197, 208, 213  
Nitrites, 267  
Nitroglycerine, 159, 192, 194, 267  
Nodding of head, 228  
Node, auriculo-ventricular, 18  
sino-auricular, 17, 25  
Tawara, 18  
Nodules, subcutaneous, 101  
Obesity, 151, 155  
Ophthalmoscopic examination, 153  
Ordinates, 70  
Orr and Innes, 157  
Osler, W., 190  
Ouabain, 266

## INDEX

- Output of heart, 27  
 Overeating, 155  
 "P" wave, 71, 76, 77, 78, 80  
     absent, 75, 76, 79  
     diphasic, 71  
     inverted, 71, 75, 77  
     in mitral stenosis, 110  
     normal, 71  
     notched, 71  
     time before contraction, 72  
 Pain, 31, 183  
     in angina pectoris, 187, 188  
     in cardiovascular syphilis, 180  
     differential diagnosis, 187  
     in hand, 188  
     in left arm, 176  
     in leg, 145  
     precordial, 101, 176  
     treatment, 253  
 Pallor around mouth, 167  
 Palpation, 33  
 Palpitation, 31, 179, 202  
 Panophthalmia, 121  
 Papillary muscles, 17, 19  
 Pardee, H. E. B., 89, 191, 260  
 Parkinson, J., 168  
 Paroxysmal tachycardia, 60, 76, 163,  
     177, 183, 201, 206  
     diagnosis, 202  
     etiology, 201  
     pathology, 201  
     prognosis, 203  
     symptoms and signs, 201  
     treatment, 203  
     ventricular type, 77  
 Passive motions, 250  
 Pause, post-systolic, 199, 210  
     compensatory, 199  
 Peabody, F. W., 48  
 Peabody, Sturgis, Thompkins, and  
     Wearn, 162  
 Percussion, 35, 38, 87  
     limitations, 35, 87  
 Pericarditis, 115  
     acute, 237  
     adhesive, 34, 112, 240  
     with effusion, 38, 101, 115  
     rub, 101  
 Pericardium, 21  
     paracentesis, 239  
     surgical drainage, 240  
 Peripheral vessels, in exercise, 27  
 Petechiae, 123  
 Phonocardiography, 88  
 Physiology, 22  
     cardiac cycle, 22, 23  
     functions of heart muscle, 27  
 Physostigmine, 197, 213  
 Pistol-shot sound (see sounds)  
 Pituitary extract, 267  
 Place, E. H., 168  
 Plesch, 38  
 Pneumonia, 31, 119, 121, 135, 142, 154  
 Pneumopericardium, 240  
 Polycythemia, 170, 233  
 Polygram (see individual waves)  
     analysis, 54, 56  
     arteriogram, respiratory variation  
         of, 52  
     artifact in, 56  
     exaggerated waves, 56, 62  
     phlebogram, 54  
 Polygraphs, types, 49  
     vibration frequency, 50  
 Polygraphy, 49, 64  
 Porter, W. T., 107  
 Position of patient (see posture)  
 Posture of patient, 38, 41, 211, 218,  
     238  
     effect of change of, 112  
     effect on rate, 202  
     of baby, 173  
 Potassium iodide, 140, 243  
     salts, 197, 208, 213  
 Potential heart disease, 113, 134  
 P-R interval, 57, 71, 80  
 Pratt, F. H., 186

- Precordia, bulging of, 32, 170  
 Premature beat (see extrasystole)  
 Preponderance, ventricular, 73, 83,  
     110, 154, 237  
     index, 73  
 Pressure curves, 24  
     intra-auricular, 107  
     intraventricular, 107  
 Price and Mackenzie, 166  
 Protodiastolic sound, 109  
 Psychic aspects, 250  
 Ptosis of heart, 47  
 Puerperal sepsis, 31, 97, 119  
 Pulmonary insufficiency, 172, 234  
     stenosis, 171, 234  
     veins, 16  
 Pulmonic valve, palpable closure of,  
     35  
 Pulsation, carotid, 32  
     epigastric, 32, 104, 237  
     expansile, 132, 242  
     second and third interspaces, 131  
     suprasternal, 131  
 Pulse, alternation (see pulsus alternans)  
     anacrotic, 60, 231  
     bigeminal, 52, 219, 220, 258  
     bisferiens, 231  
     capillary, 228  
     Corrigan, 105, 136, 173, 227, 229  
     deficit, 209, 210, 212  
     paradoxical, 53, 112  
     plateau, 231  
     pseudo-alternation, 52, 219  
     respiratory variation in, 52  
     tracings (see polygram)  
     venous, 234  
         auricular form, 57  
         inspection of, 211, 215  
         ventricular form, 57, 61, 210  
         wave, velocity of, 230  
 Pulsus alternans, 52, 219  
 Pulsus paradoxus (Kussmaul's pulse),  
     53, 112  
 Pupils, unequal, 131  
 Purkinje fibres, 19  
 Pustules, 120  
 "Q" Wave, 71  
 QRS complex, 69, 71, 82  
 Quinidine, 207, 213, 268  
 "R" wave, 71, 73, 74  
     notched, 72  
     prolonged, 72, 82  
     thickened, 72  
     time before contraction, 72  
 Rash on skin, 120, 122  
 Rate, 28, 62, 78, 81, 202, 210, 215, 270  
     acceleration, 198, 200  
     relation to arrhythmias, 199  
     doubling of, 196  
     halving of, 196  
     rule of, 211  
 Ray fish, 205  
 Reid, W. D., 25, 89, 108, 111, 126, 127,  
     135, 138  
 Renal function, 152, 154  
 Respiration, paralysis, 271  
     whistling, 131  
 Rest, 198  
     in bed, 115, 140, 148, 169, 248  
 Restlessness, 253  
 Resuscitation of heart, 256  
 Retraction, of cardiac impulse, 32,  
     104  
     of chest wall, 111  
 Reuta, 126  
 Rheumatic fever, 19, 97, 119, 135, 185  
 Rheumatic heart disease, 97, 136  
     Aschoff bodies, 98  
     diagnosis, 113  
     etiology, 97  
     pathology, 98  
     prognosis, 114  
     symptoms, 99  
     treatment, 115

- Rhythm, coupled, 258  
 dominant, 53, 59, 62  
 gallop, 35, 167, 215  
 idio-ventricular, 81  
 two to one, etc., 206
- Robinson and Hermann, 77  
 Robinson and Wilson, 258  
 Roll in mitral stenosis, 108  
 Röntgen findings, 38, 46, 90, 110, 133, 145, 231, 239  
 comparison with percussion, 37
- Rotch's sign, 238
- Rumble in mitral stenosis, 108
- Rupture of heart, 240
- "S" wave, 71
- Sallowness of complexion, 123
- Salt, 150, 156, 255
- Scarlet fever, 31, 97, 135, 160
- Schmorl, 126
- Schneider, E. C., 253
- Sears' point, 240
- Sedatives, 158, 200, 204
- Septic heart disease, 119, 135  
 acute type, 120  
 chronic type, 121  
 diagnosis, 123  
 etiology, 119  
 pathology, 119, 125  
 prognosis, 124  
 symptoms, 120  
 treatment, 124
- Septicemia, 124
- Septum, patent interventricular, 171, 173
- Serum therapy, 125
- Shattuck, G. C., 35
- Shortness of breath, 31, 183
- Sign, Bamberger's 288  
 Broadbent's, 112  
 Duroziez's, 229  
 Ewart's, 238  
 Litten's, 112  
 Rotch's, 238
- Sino-auricular block, 58, 75, 196
- Sinus arrhythmia, 53, 57, 74, 195  
 phasic type, 58, 74
- Sinus of Valsalva, 17, 20
- Sinus venosus, 15
- Size of heart, 36, 46, 86, 93, 202, 236  
 cardio-thoracic ratio, 46, 90
- Skin, petechiae, 123  
 pustules, 120  
 rashes, 120, 122  
 syphilitic lesions, 134
- Small-pox, 135
- Smith, S. C., 167
- Sodium nitrite, 194
- "Soldier's heart" (see effort syndrome)
- Sounds, 25, 41, 88  
 aortic, 88  
 auricular, 199, 206, 207, 215  
 first, 25, 72, 88, 108  
 first, split, 88, 176  
 pistol-shot, 228  
 protodiastolic, 109  
 pulmonic, 88  
 second, 25, 72, 88, 108  
 accentuated, 88  
 aortic, 132, 146, 231  
 reduplicated, 89, 109  
 split, 88, 176  
 third, 57, 89, 109  
 timing of, 34  
 Traube's, 229
- Southey's tubes, 255
- Space, deep cardiac, 86  
 superficial cardiac, 86
- Spartein, 267
- Spas, 252
- Spleen, enlarged, 120  
 infarct, 122
- Squill, 265
- Standstill, cardiac, 256  
 ventricular, 214
- Starling, E. H., 24, 43, 66, 107, 228
- Steell, Graham, 109, 111

- Stethoscope, 39, 227,  
Stewart, H. A., 229  
Strophanthin, 197, 208, 218, 265, 266  
Strophanthus, 265  
Structural lesions, 225  
Strychnine, 267  
Sweats, 122  
Symptoms, 31  
Syphilis, 185  
Syphilis, cardiovascular, 126, 208, 218,  
    242  
    blood pressure in, 133  
congenital, 127  
diagnosis, 134  
etiology, 126  
frequency of, 126  
mitral stenosis, 103  
pathology, 127  
physical signs, 131  
prognosis, 137  
symptoms, 130  
therapeutic test, 135  
treatment, 138
- "T" wave, 72, 162, 258  
    flattened, 72  
    inverted, 72  
    normal, 72  
Tabes dorsalis, 137  
Tachycardia, differential diagnosis,  
    177  
    effort syndrome, 168, 177  
    hyperthyroidism, 161  
    (see paroxysmal tachycardia)  
    regular, 202  
Tea, 198  
Teeth, 97, 117, 188  
Thayer, W. S., 89  
Thayer and MacCallum, 172, 229  
Thebesius, veins of, 20, 186  
Theobromin sodium salicylate, 155,  
    268  
Theocin, 155, 266, 268  
Theophyllin, 197  
Theophyllin sodioacetate, 155, 268  
Therapeutic test, 135  
Thrill, 34, 85, 231  
    at apex, 105  
    at base, 132, 172, 173, 174  
Thrombosis, 110, 166  
Thyroid heart (see hyperthyroidism)  
Time marker, 55, 70  
Tobacco, 190 (see nicotine)  
Tonsillectomy, 117, 248  
Tonsillitis, 31, 97, 119  
Tracheal tug, 242  
Transfusion of blood, 125  
Traube's sounds, 229  
Treatment, drug, 257  
    etiological, 247  
    exercise, 118  
    general, 248  
    regulation of life, 118  
    symptomatic, 118, 253  
Tricuspid insufficiency, 233, 234  
    relative, 234  
Tricuspid stenosis, 233  
Tuberculosis, 135  
    of heart, 114, 238  
    miliary, 128  
    pulmonary, 137, 170  
Typhoid fever, 128, 185, 142
- "U" wave, 72
- "V" wave, 55
- Valves, 16  
Vaquez and Bordet, 202  
Veins, anterior cardiac, 20  
    cervical, 32  
        diastolic collapse, 112  
        engorgement, 181, 184  
        inspection of, 211, 215  
    pulmonary, 16  
        of Thebesius, 20, 186
- Venesection, 158, 254  
    "bloodless," 255

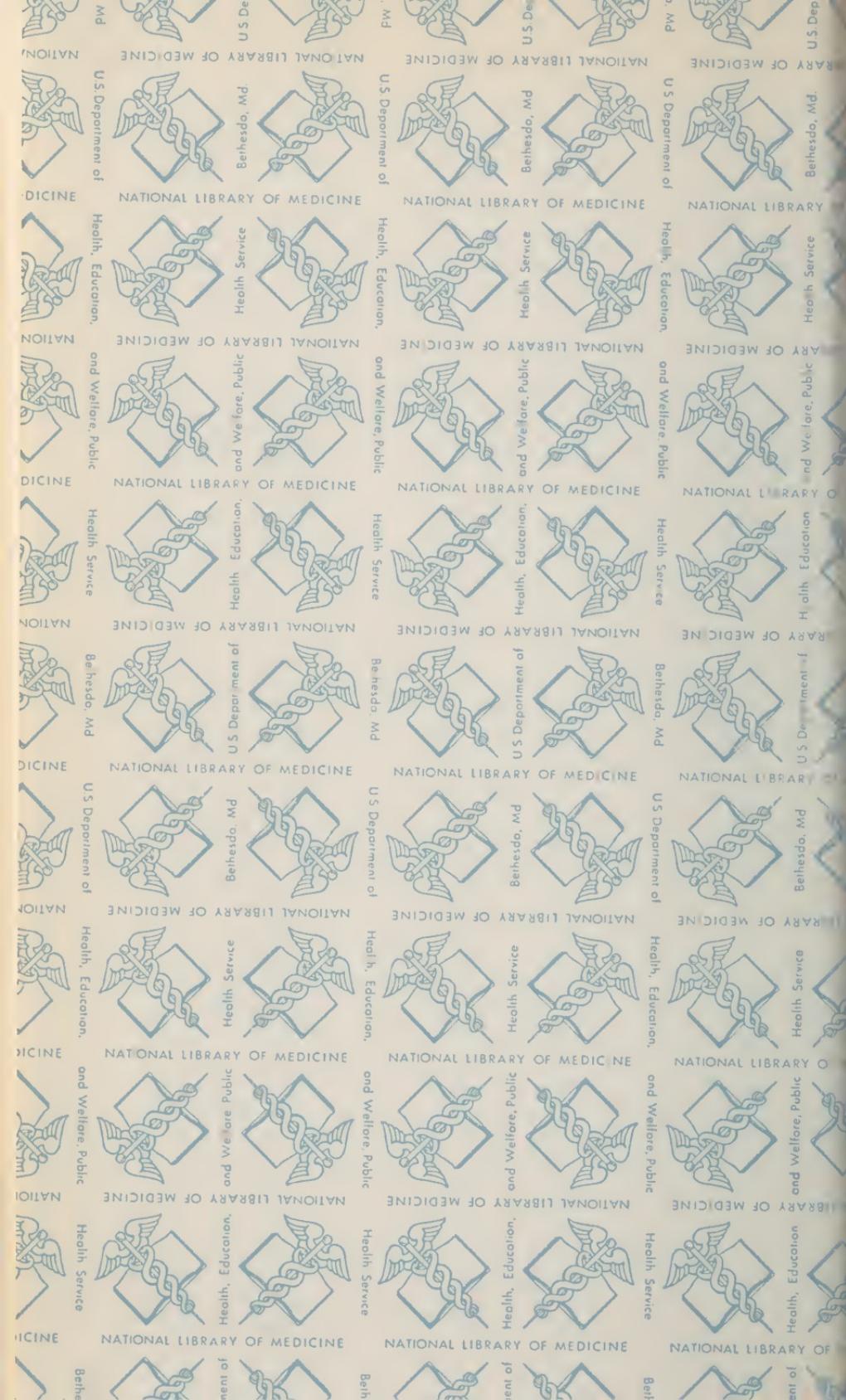
## INDEX

- Ventricle, left, 16  
right, 16  
Ventricular escape, 75, 80, 258  
filling, 107  
output, 27  
systole, 23, 25  
Veronal, 200, 204  
Vital capacity, 47  
Vomiting, 169, 203, 204  
  
Walker, R. W. S., 173, 227  
Waller, A. D., 66  
Walton, G. L. W., 158  
Warthin, A. S., 126, 127, 129  
Wassermann reaction, 133  
  
Water (see fluid intake)  
amount of, 157  
Weiss and Hatcher, 265  
Wenkebach, 49  
White, B., 125  
White, P. D., 219, 265  
White and Aub, 162  
White and Bock, 73  
Wiggers, C. J., 25, 50, 229  
Williams, 67  
Williamson, C. S., 238  
Wilson and Hermann, 72, 73, 83, 237  
Worry, 151, 158  
  
Zollinger, F., 229











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